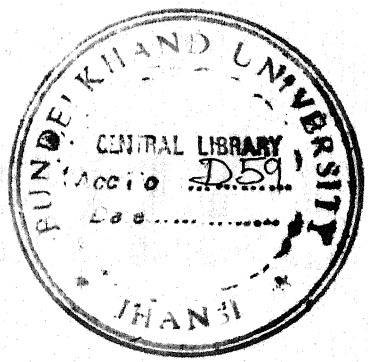
"PLACENTAL GRADING, AMNIOTIC FLUID INDEX AND FETAL OUTCOME IN IUGR"

THESIS FOR

DOCTOR OF MEDICINE

(OBSTETRICS AND GYNAECOLOGY)



BUNDELKHAND UNIVERSITY

JHANSI (U.P.)

2005 DR. MANJU MAHAR

Dedicated

to

My Parents

CERTIFICATE

This is to certify that the work entitled ***"Placental Grading, amniotic fluid index and fetal outcome in IUGR"*** which is being submitted as a thesis for ***MD (Obstetrics and Gynaecology)*** examination, 2005 under Bundelkhand University by ***Dr. Manju Mahar*** has been carried out in the ***Department of Obstetrics and Gynaecology, M.L.B. Medical College, Jhansi*** under my direct supervision and guidance. The observations recorded have been checked and verified by me from time to time.

She has put in the necessary stay in the Department as per required by the regulation of Bundelkhand University.

Dated: 13/8/05

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Jhansi

Dated: 12/5/05

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Introduction

INTRODUCTION

The desire of every woman contemplating motherhood is that her pregnancy culminates in a healthy offspring who will achieve the highest possible physical and mental potential. Towards achieving this goal it remains the obstetricians responsibility to reduce the well-recognized implications and consequences of intrauterine growth retardation by early diagnosis and management.

The growth-retarded fetus represents a failure to achieve recognized normal growth parameters.

There is a deceptive simplicity about the diagnosis of fetal growth retardation and there is no reliable definition of IUGR available before birth admittedly it can only be made in retrospect, when in utero becomes ex utero, and you can weigh the baby. The most commonly accepted definition being babies weighing less than the 10^{th} percentile for their gestational age at birth.

Intrauterine growth retardation complicates 10% of all pregnancies and is associated with 6 to 8 fold increase in perinatal mortality and morbidity. Fetal demise, birth asphyxia, meconium aspiration and neonatal. Hypoglycemia and hypothermia are all increased, as is the prevalence of abnormal neurological development. This is true for both term and preterm infants. Postnatal growth and development of the growth restricted fetus depends on the cause of restriction, nutrition in infancy, and the social environment. Infant with growth restriction due to congenital viral, chromosomal or maternal constitutional factors remain small throughout life. Those infants with inutero growth restriction due to placental insufficiency will often have catch up growth after

birth and approach their inherited growth potential when provided with an optimal environment. Similarly, neuro developmental outcome of the growth-restricted fetus is influenced by postnatal environment.

Direct fetal visualization in utero by ultrasonics was introduced by **Donald and Brown** in 1960. Since then the use of ultrasound to approximate the gestation age of the fetus and assess its well being in utero has become wide spread. Fetal biparietal diameter was the first parameter to be measured for this diagnosis. Subsequently continuous attempts were made to discover a more accurate parameter and various ultrasound-measured indices were used either singly or in formulations. These included abdominal circumference, thoracic circumference, head circum ference, crown rump length and femur length, cross sectional areas of head and abdomen, quantitative assessment of liquor and total intrauterine volume and echogenecity of placental tissue.

Changes in placental texture and structure on ultrasonic examination have been suggested as correlating to advancing fetal pulmonary maturity. The association of preterm appearance of grade III changes in the placenta in the complicated pregnancy suggests that these changes are associated with a decrease in placental function and premature senescence of the placenta. Premature placental maturation grade III has been associated most commonly with pregnancy complicated with intrauterine growth retardation. **Grannum et al** introduced this concept in 1983. The association of premature appearance of grade III changes in the placenta in pregnancies complicated by maternal diseases known to produce a decline in placental function suggests that incidental finding of grade III placental changes at the time of ultrasound may indicate unrecognized pregnancy complications.

The morphologic changes that occur with placental senescence can be observed by ultrasound and have been described by **Grannum, Berkowitz and Hobbins.**

Grade 0:- During the first part of gestation, the ultrasonic appearance of the placenta is homogeneous, without echogenic densities, and limited by a smooth chorionic plate.

Grade I:- With progression of pregnancy, the chorionic plate begins to acquire subtle undulations, and echogenic densities appear randomly dispersed throughout the organ but spare its basal layer.

Grade II:- Near term the indentations in the chorionic plate become more marked, echogenic densities appear in the basal layer, and commalike densities seem to extend from the chorionic plate into the substance of the placenta.

Grade III:- Finally, when the pregnancy is at term or post term, the indentations in the chorionic plate become more marked giving the appearance of cotyledons. This impression is reinforced by increased confluency of comma like densities that becomes the intercotyledonary septations. Also characteristically, the central portion of the cotyledons become echo-free (fall out areas) and large irregular densities capable of casting acoustic shadows, appear in the substance of the placenta.

The correlation between ultrasonic signs of placental senescence and the functional capacity of the placenta is poor. The correlation between grade III placenta and fetal pulmonary maturity is excellent in pregnancies near term.

Clinical assessment of amniotic fluid volume is an important part of foetus assessment as variation in its amount have been related to a variety of pregnancy complications. Amniotic fluid volume provides important information about foetus renal and placental functions.

Even though amniotic fluid is ultimately derived from the mother the physiological processes involved in its production are still not clear and investigation is complicated by the multiple possible sites of formation. The fluid could arise as a secretion or ultra filtrate from the mother across the membranes, from the placenta cord or from the foetus through the skin, Git, tracheobronchial tree or kidneys.

The methods used in the past for estimating the volume of liquor amnii were crude and invasive. With the advent of ultra sonography for antepartum surveillance has come a movement away from the previous invasive techniques and has allowed a more complete evaluation of the foetus and its intrauterine environment.

Amniotic fluid assessment is a tool for evaluation of foetus well being. Because of the uterine cavity being irregular, direct evaluation is difficult and subsequently indirect techniques have been utilized.

Evaluation of amniotic fluid volume is an important aspect of obstetrical ultrasound. Several methods are currently used to describe amniotic fluid volumes, the most common are the subjective assessment of the fluid and the semiquantitative methods. The reproducibility of these methods is excellent in experienced hands.

Abnormal amniotic fluid volume may be the only or earlist sonographic sign of obstetrical problem. Therefore it is important that sonologist and obstetrician are familiar with amniotic fluid volume assessment.

A borderline amniotic fluid index observed in antepartum testing is associated with an increased risk of intra uterine growth restriction and overall adverse perinatal outcome. This finding is thought to be a result of decreased perfusion of the fetal kidneys, thereby resulting in decreased urine production. Oligohydramnios is present in the majority of IUGR infants (Approximately 80 – 90%) but the presence of a normal amniotic fluid index (AFI) should not preclude the diagnosis of IUGR. Amniotic fluid volume should also be assessed atleast weekly in at risk pregnancies, because the likelihood of a fetus being small because of nutritional deprivation is much less when normal amniotic fluid volume is present.

Three different techniques of ultrasonographic assessment of amniotic fluid volume have been reported.

1. Simple visual estimation with the relationship of intrauterine contents.
2. Vertical measurement of the single maximal amniotic fluid pocket.
3. The four-quadrant summation of the maximal amniotic fluid pocket or the AFI.

In the present study for the measurement of amniotic fluid index time ultrasound scanning has been performed as described by **Phelen et al.** A 4 quadrant amniotic fluid volume is assessed by placing the ultrasound transducers perpendicular to the wall of uterus and parallel to the mother's spine in four abdominal quadrants. Pockets consisting primarily of umbilical cord are discarded. A four quadrant sum of 8.1 – 18cm is considered normal.

Aims & Objectives

AIMS AND OBJECTIVES

- To study the association of placental grade with birth weight in IUGR.
- To study the correlation of placental grade with perinatal outcome in IUGR.
- To study the difference in AFI values in normal pregnancies and pregnancies complicated with IUGR.
- To study the correlation of amniotic fluid index with perinatal outcome in IUGR.

Review of Literature

REVIEW OF LITERATURE

Fetal growth retardation ranks third after prematurity and malformation as a cause of prenatal deaths. Antenatal fetal monitoring has emerged as the most important means of reduction in the number of still births and improvement in the quality of survival of infants who are born alive. Clinical acumen combined with biochemical and ultrasonographic testing will identify as many as 70% of growth-retarded fetuses. It should be the obstetrician's aim to identify all growth retarded fetuses at risk of death from hypoxia.

In 1947, **Macburney** first introduced the concept of IUGR. **Clifford** in 1954, actually claimed that clinically significant intrauterine malnourishment can occur in utero associated with intrauterine asphyxia and soft tissue wasting and he attributed these changes to the decreasing placental function with advancing maturity. **Karn and Penrose** (1951) showed that the weight at which the mortality curve reaches its minimum is several hundred grams heavier than the mean birth weight, a finding which was true for all human population later investigated (**Wilcox and Russill**, 1983)

Subsequently many workers entered into this field and much research has been done into the etiology, pathology, diagnosis and management of this condition, its immediate and long-term effects on the fetus and neonate. Much confusion has occurred by inundating IUGR, with a number of synonyms ..... 'small for gestational age' (SGA), 'small for date' 'dysmature', 'Low birth weight', 'placental insufficiencies', all being basically used to refer to the growth retardation.

The most widely accepted definition is that given by **Lubchenko** 1963, **Battaglia** 1966; **Bard** 1970; **Fedrich and Adelstein,** 1978 ..... a foetus whose birth weight is below the 10^{th} percentile for that period of gestation.

Types of growth retardation:

The classical picture of the growth retarded infant – increased body length in relation to weight, relatively large head with wide skull sutures, muscle wasting prominent ribs, an alert look and a dry wrinkled skin, is readily recognized. Reduction in length and in brain weight is much less than the fall in body weight where as weight of liver and spleen are relatively more reduced than body weight. In the past with unreliable data on gestational age, such disproportion mainly expressed in terms of birth weight compared with length and / or head circum ference were important criteria to distinguish preterm infants from growth retarded infant, (**Gruehwald,** 1963)

In general, these clinical types of IUGR can be subdivided into a soft tissue type (In which there is reduced soft tissue mass, mainly adipose tissue and muscle wasting; a skeletal type (In which length and head circumference are more affected), and a combined type which shows features of both the skeletal and soft tissue type. Various term have been used by investigators to indicate differences between the first two types. One thus encounters subdivision of subacute versus chronic, symmetric versus asymmetrical, disproportional versus proportional, wasted versus symmetrically small, soft tissue wasting versus underweight for gestational age, low ponderal index versus short for dates, and so on.

Distinction between disproportionally and proportionally grown or growth retarded infants is usually made on the basis of the ponderal index, a

measure introduced some 60 years ago by Roghrer (1921) for comparing nutritional status of infants. This index .....the product of (birth weight in g) X 100 / (Length in cm)3, shows how heavy the infant is for his length and increases with accumulation of muscle mass and adipose tissue.

Ultrasonic assessment of fetal growth has revealed two principal types of growth retardation (**Campbell,** 1974). The first, called late flattening can be detected by serial measurements of biparietal diameter, the BPD is under the normal range until after 30 weeks of gestation when its rate of growth slows or stops. Generally retardation of growth of trunk is affected earlier and more severely. Comparisons of head and trunk growth have revealed a disproportionate decrease in the latter in 84.2% and 100% of such cases in studies of 19 & 7 fetuses respectively. (**Campbell S. Thums A** 1977). Thus, disproportion is a common feature and therefore also the term asymmetric growth retardation. Basically this type of IUGR is due to factors, which compromise nutrition in an otherwise potentially healthy infant. There is a brain affect and there is preferential supply of fetal circulation to head and brain over the trunk. A second type of growth retardation is called low profile. This growth pattern is characterized by a BPD that grows consistently slower than normal at least after 2 weeks. The fetus exhibits growth retardation which begins in 2nd trimester and affects all parts of the body more or less uniformly. Campbell considered it a manifestation of reduced growth potential and has also referred to it as hypoplastlc IUGR, as the total number of cells are reduced. The low profile child has a relatively trouble free delivery, but later it is found to be of subnormal IQ and stature (**Fancourt et al,** 1976). Affected fetuses are at much greater risk for congenital malformations than the general population (**Ramzin et al** 1973). According to **Hansman** (1976) a significant number of these fetuses suffered intrauterine infections.

Aetlology:

Fetal growth can be defined as time dependent increases in specific geometrical characteristics of the fetus and it depends on a complex interplay of fetal, maternal and placental factors. Malfunction of any of these factors lead to growth retardation. Thus, IUGR is multifactorial in origin and it is important to note that in over half the cases no obvious cause can be implicated potentially.

Potential causes of IUGR are dealt with below:-

Chromosomal abnormalities are estimated to occur in about 6% of conceptions (**Weight**, 1976) and in 5% of recognized conceptouses (**Hood,** 1981).

The presence of multiple congenital malformations often leads to a clinical diagnosis of IUGR. **Swaab et al** (1978) found that at 40 weeks of gestation the mean anencephalic birth weight was about 1000g, less than mean normal birth weight minus the weight of the brain. Of the congenital deformities those which affect the central nervous system and / or skeletal system have the most marked effect on fetal growth. IUGR is also common in fetuses with gastrointestinal abnormalities such as duodenal atresia (**Girvans and Stephen** 1974); omphalocoele (**Columbani and Cunningham**, 1977). Potter's syndrome and renal agenesis too are associated with IUGR.

IUGR may be caused by infections of viral or bacterial origin. Upto 60% of infants with congenital rubella may be below the 10^{th} percentile of weight for gestation (**Coops et al,** 1965). The rubella virus shows a predilection for infecting vascular endothelium and may cause placental villous by inducing in the endothelium of villous capillaries (**Driscoll**, 1969).

C.M.V. is currently a common cause of congenital viral infection with an incidence ranging from 0.2% to 2.2% in different population (**Stanzo et al,** 1983). Growth retardation occurs in about 40% of infants who present with clinical manifestations at birth (Stanzo et al,1983). Varicella zoster infection especially early in pregnancy can cause IUGR (**Waterson and Lynel,**1947). Growth retardation is common in malarious mothers. In an endemic malarious area **Mcgregor, Wilson and Billsicz** (1983) found that 20% of placenta were infected and in these cases birth weight was reduced by about 170g.

According to **Brent and Jensh** (1967) high dosage ionising radiation during pregnancy may result in severe growth retardation. Several drugs have been reported to cause impaired fetal growth (**Jhons and Chernoff,** 1978). Howard and hill 1979, Redmond 1979). Certain drugs appear to affect some but not all parameters or growth for eg. Hulesmaa et al (1981) found smaller head circumference without much changes. In other parameters with use of the antiepileptic carba-mazepine or with a combination of phenobarbitone and phenytoin, chronic administration of corticosteroid are possible causes of growth retardation (**Scott,** 1977). More than 50 publications based on over ½ a million births reported that a woman who smoked during pregnancy had babies of lower birth weight than woman who did not. The size of differences (above 175 to 200 gm or a depression of approximate 5%) was remarkably consistent in all investigations (**Peter, et al,** 1983). Effect of alcohol abuse on fetal growth may be mediated in a number of ways, direct effect of alcohol or its metabolite acetaldehyde are probably the most important. These include malabsorption of nutrients across the intestinal mucosa, alleged maternal hepatic function, effects on amino acid transport across the placenta and effects on fetal metabolism and endocrine function (Rrosett et al, 1983). Reduced fetal growth is most marked in the fetal alcohol syndrome which is characterized by.

(a) Prenatal growth retardation

(b) Congenital malformation

(c) Facial dysmorphology

(d) Disturbances of mental development.

Narcotics alone can impair growth (Renenteria and Lotongknum, 1977). Amongst infants exposed to heroin in utero as many as 50% are growth retarded.

Placental influences:

In the absence of gross pathology of the maternal fetal unit placental size shows a close correlation to fetal size. A large placenta is required to produce in large baby but the reverse is not necessarily true. Nevertheless, most growth retarded fetuses, have a fetal placental weight ratio which is higher than that of normally grown fetuses. Thompson, Billewiez and Hitter (1968) showed that the fetal placental weight ratio increased from approximately 4.5 in the higher placental weight groups to about 7.3 in the lowest I e a growth retarded foetus shows some compensatory growth and tends to outgrow its small placenta. Of the many macroscopically observed placental alterations (for e-g. calcification or so called infants that have been held responsible for otherwise unexplained fetal growth retardation, few have stood up to close scrutiny (Fox, 1981). Placental abnormalities truly associated with low birth weight for gestation are haemangiomata and extrachorial placenta (Fox, 1981).

Maternal vascular disease:

There appears to be general agreement that vascular pathology whether it is due to renal disease, essential hypertension, PIH, diabetes or collagen vascular disease, is the single most common denominator in the causation of

IUGR. Frequency and severity of growth retardation is highest in pregnancy induced hypertension superimposet on preexisting hypertensive disorders. Lastly in PIH there is an undeniable relationship with gestational age and the onset of disease. **More and Fedman** (1983) found that 82% of infants of mothers in whom they diagnosed preeclampsia before 34 weeks of gestation had birth weight below the 10^{th} percentile of weight for gestation.

Anaemia and low haemoglobin levels, vary with poor socioeconomic status, poor general nutrition and other factors known to be associated with poor birth weight (**Butler and Alberman**, 1964) **Harison and Ibeziako** (1973) demonstrated clearly that severe chronic anaemia (Defined as haematocrit of less than 30%), was associated with a reduction in birth weight of about 100 gm, per 2% packed cell volume.

Perinatal Mortality and Morbidity and Long Term Sequelae Associated with IUGR:

20% of all fetal deaths can be attributed to the impact of this problem and is reflected in a marked increase in perinatal mortality and morbidity associated with IUGR (Batteaglia, 1970; Dobson et al 1981; William et al 1982). It is associated with an eight fold increase in mortality at birth due to anoxia, asphyxia, and decreased PH and a significant increase in the incidence of neonatal problems such as polycythaemia, hypothermia, hypocalcaemia.

The role of ultra sound in the evaluation of IUGR:

Since the introduction of pulsed echo ultrasound into obstetric diagnosis in 1958 (**Donald, Macvicar and Brown,** 1958). Ultrasonic diagnosis is concerned with the diagnosis of pregnancy, the viability and normality of the fetus, and in particular the accurate establishment of gestational age. In the latter half of pregnancy the major use of ultrasound is

in the assessment of fetal growth, which is the outcome of complex interactions between the fetal genotype and numerous constraining and growth accelerating factors with in the fetomaternal environment.

Amniotic Fluid:

It was **Hippocrates** (460 – 377 BC) who suggested the oldest and simplest explanations that the amniotic fluid is a product of foetal kidney.

Portal (1985) supported this contention which was accepted in general till 19th century which marks a remarkable turning point in the history of medicine by way of better understanding of physiology and physiological chemistry of the fundamentals of human biology.

Prochowrik (1877): Furnished data on water solid ash, urea and chloride of amniotic fluid. This started the doubt on the time wondered sea water bath concept for liquor amnii which Picturesque could no longer explain the complete nature of amniotic fluid in its entirety.

The belief prevailing in France that liquor amnii was an accumulation of menstrual blood or milk was changed by **Francois Mauriceu** (1637 – 1709) **Fourcroy** (1970 – 1803) considered amniotic fluid as arising from embryonic and maternal blood with contribution from fetal urine. However in two accidental cases of sulphuric acid poisoning to mother the acid was detected in liquor amnii while none could be detected in foetal urine (**Gusserow** 1872). This supported the view that amniotic fluid arose from maternal blood. (Zuntz 1878 zuntz and weiner 1881): Their experiment supported the view that amniotic fluid arose from the maternal blood.

Schallar (1899): Injected phloridizine into mother, the sugar appeared in the foetal urine but not in the amniotic fluid. On the contrary increased incidence of polyhydramnios in cases of chorioangioma and uniovular twins (**Kraus** 1903) and stoppage of amniotic fluid secretion after death of foetus in utero even though maternal part of placenta remained normal for some time (**Watson** 1906) lent support to the belief that the amniotic fluid was foetal in origin.

Albano and Politi (1903): Observed that liquor amnii was hypotonic to maternal and foetus serum in many animals. While Zange Meister and Meisse from similar observation in human amniotic fluid hypothesized that dilution of amniotic fluid by foetal urine was responsible for such change.

Lsne and Binet (1921): States that bladder of the new born always contains 5 – 8 cc of urine. **Taussig** (1925) states that in his cases absence of foetal kidneys or blocking or foetus urinary passage led to oligohydramnios. It is also true that normal amount of fluid and even hydramnios have been found to be associated with the same diseases.

As a result of injecting sodium phenol sulphonapthalate into the amniotic cavity, **Albano** (1934) suggested that the amniotic fluid in the ovum could be changed through the maternal circulation. However, it was largely due to the classical isotope studies of vosburgh et al (1948) that the fluid interchange between the liquor and the fluid compartments of the foetus and mother was demonstrated.

Setnikar Agostoni and Taglaietti (1959): Have showed experimental evidence in goats and guinea pig for the production of liquor amnii by the foetus lung at least in the later part of pregnancy.

The demonstration both in vitro: Parmley and seeds – 1970 Lind et al (1972) and in vivo (Abramovich and Page – 1972) that the permeabicity of the foetal skin differs markedly during pregnancy increase the possible variables in the production, circulation and fate of the amniotic fluid at the different stages of gestation.

There is immunological evidence from the study of protein solute that both the foetus and the mother make individual contribution to the amniotic fluid (Sutcliffe, 1975).

The role of Placenta and membranes:

Fluid is present in the case of some blighted ova where the foetus is rudimentary or absent. **Jeffcoate and Scott** (1959): These facts suggest that, initially anyway, amniotic fluid enters via the membranes.

Further support came from **Berhman et al** (1967) who showed that when the foetus was surgically removed from the monkey uterus a limited further quantity of liquor was formed.

The role of direct transport across the membranes was investigated by Danfurth and Hull (1958) who examined living amniotic epithelium using phase contrast microscopy and demonstrated features they considered to be typical of secretory cells. However, these amniotic cells degenerate most rapidly and plant (1966) has shown that polyhydramnios may be associated with a complete absence of these cells.

The brush border, demonstrated by **Taussig** (1927) using light microscopy and later confirmed by **Bourne** (1962) with the electron

microscope, could suggest secretary function. In the human foetus there is evidence that there is no electrical potential difference across the membranes (**Mellor et al** 1969) suggesting that only passive diffusion takes place.

Abramovich and Page (1972): Believe there is a specialized area of that part of the amnion overlying the placenta which, because of its extensive vascular connections, plays a greater part in fluid exchange. The association of polyhydramnios with hemangiomatous tumors of the chorion could be interpreted as evidence of increased liquor production at this site, though an alternative explanations is that retention of foetal metabolites results from partial obliteration of the chorion decidual space resulting in increased foetal urinary production (**Mcinruy and Kelsey,** 1954). Oligohydramnios is associated with the condition of amnion nodosum where the amniotic epithelium is histologically deranged and **Landing** (1950) felt that this represented a loss of secretory function.

Jeff Coate and Scott (1958): Described 14 cases of amnion nodosum of which 10 had foetus renal agenesis and the other all had oligohydramnios explainable on other grounds. They believed that the historical changes were the result rather than the cause of the reduced liquor volume.

The role of the foetus and cord:

Lind (1957) Has shown that if the more diffusible solutes are examined the amniotic fluid most closely resembles extension of the foetal extra cellular space in the 1st half of pregnancy modified later by the loss of foetus skin permeability accompanied by increasing intrauterine foetal organ function. He also calculated that the volume of liquor in early pregnancy was related most directly to the weight and, hence to the surface area of the foetus rather than the placental weight or length of gestation.

The foetal skin has been shown to be permeable to fluids and to some dissolved solutes upto 18^{th} weeks of pregnancy, after which increasing keratinisation and skin thickness markedly reduces this route of access.

Abramovich and Page (1972) used radio markers in human foetus at the time of termination and confirmed the permeability of skin in mid trimester foetus but believed that the exchange take place through the whole feto placental unit of foetus, cord and placental plate and suggested that at the 18^{th} to 20^{th} week the cord takes over as the major transfer site.

Observations by Thomas et al (1963), comes from radiographic confirmation that radio opaque dye given to the mother through an intra arterial catheter for placental localization is concentrated by the foetal kidney resulting in intrauterine foetus pyelogram.

The increasing biochemical resemblance of liquor to foetal urine, particularly after keratinisation of the foetus skin, is seen by increasing concentration of urea, uric acid, creatinine increasing sodium and glucose and a reduction in osmolality. Poulsen 1955, Lind 1975, Pitkin and Reynolds 1975, young 1976, Campbell et al (1975): visualized the foetal bladder using ultrasound screening and demonstrated in utero bladder emptying.

The association of fetuses with bilateral renal agenesis and oligohydramnios has been recognized for some time (Atopsasion, 1936; Green 1955; wagner and oygstrup, 1963; Potter 1965) from this association it has been confirmed that the absence of urinary output by the foetus results in the failure of amniotic fluid formation. The role of the foetal respiratory tract in the formation of liquor is much less fully substantiated.

The relatively high chloride content of amniotic fluid has been attributed to pulmonary secretion (Goodlin and Rudolph, 1970), though the origin might alternatively be from the foetal stomach (Lind, 1975).

The most convincing explanation on the evidence available at present is that intrauterine breathing movements result in a small tidal flow of liquor (Whitfield, 1976), thus allowing absorption by the lung and at the same time access for pulmonary secretions to the amniotic fluid. The precise origin of liquor amnii still remains unsolved, it is probably of mixed maternal and foetal origin. The following are the speculative theories:

1. As a transudate from maternal serum across the foetus membranes or from the maternal circulation in the placenta.
2. As a transudate across the umbilical cord or from foetus circulation in the placenta or secretion from amniotic epithelium.
3. Contribution from foetal urine the foetus drinks about 400ml or liquor every day at term and equal to that is excreted in urine, if either ingestion or micturition is seriously interfered with, gross alteration of volume occur.
4. Secretion from the tracheobronchial tree and across the foetal skin before the skin becomes keratinised at 20^{th} weeks.

The liquor volume previous investigations have shown a progressive rise in the amniotic fluid volume during pregnancy. Wagner and Fuchs in 1962 were able to quantitate at the time of hysterotomy and abortion a progressive increase in the amniotic pluid volume from 10 – 20 weeks.

Gadd et al 1966 in subsequent publication were able to corroborate their work using dye dilution technique, he demonstrated a progressive rise in

the amniotic fluid volume from early 2nd trimester that reaches the zenith in the early 3rd trimester. There after amniotic fluid volume remained at a plateau until 38 weeks gestation, then it gradually fell.

Beisher and associates in 1969: Demonstrated in a group of postdated patients that amniotic fluid volume underwent a rapid decline during this critical period of pregnancy. **Gohari** and associates in 1977 used contact B san were able to show a progressive rise in the total intrauterine volume throughout pregnancy.

The use of this technique enabled them to identify the foetus with the greatest probability of IUGR. Thus amniotic fluid measures about 50ml at 12 weeks 400ml at 20 weeks, and reaches its peak of 1000ml at 36 – 38 weeks thereafter the amount diminishes and at term measures about 600 – 800ml as pregnancy continues post term, further reduction in amount occurs.

Ultrasound evaluation of amniotic fluid volume:

The advent of ultrasonography has come a movement away from previous invasive technique of assessing amniotic fluid volume.

Gohari and associates (1977): Using contact B scan ultrasonography moved a progressive rise in the total intrauterine volume throughout pregnancy. Ultrasonographic estimation of amniotic fluid volume is an important adjunct to assessment of foetal well being. However no single method has emerged predictive. Subjective scales have been proposed but they lack reproducibility.

Several sonographic techniques designed to evaluate amniotic fluid volume have been reported in the literature. **Philipson EH sokot RJ**

Williams in oligohydramnios – clinical association and predictive value for IUGR (1983) have mentioned three criteria for oligohydramnios.

1. Obvious lack of amniotic fluid
2. Poor fluid / foetal interface.
3. Marked crowding of foetal small parts.

Maximum vertical pocket depths:

Manning et al (1980) demonstrated that absence of a single pocket of amniotic fluid 1cm in depth was associated with increased risk of foetal distress and perinatal mortality.

Chamberlain et al (1984) used a 2cm rule to define marginal amniotic fluid IUGR and perinatal death were increased in these pregnancies. Similarly a single pocket exceeding 8 cm was predictive of increased rates of macrosomia. **Hoddick et al** (1989) subsequently. Demonstrated that the 1cm rule has poor sensitivity in prediction of IUGR.

Bottoms et al (1984) noted that the maximum vertical pocket varied significantly during gestation and asserted that application of a simple depth criteria was no better than subjective grading of amniotic fluid volume. The above technique also does not permit the clinician to perform a total evaluation of the fluid within the intrauterine cavity and to follow the progressive changes in amniotic fluid volume during pregnancy.

To expand the sampling field **Phelan et al** (1987) – summed vertical fluid pockets in four quadrants of uterus the amniotic fluid index. AFI has been shown to correlate well with perinatal outcome.

AFI was found to rise progressively from 13 weeks of gestation until 26 weeks. From 26 – 38 weeks, the AFI demonstrated wide variation and peak AFI appeared at 29 – 30 weeks. After 38 weeks the AFI declined progressively. Furthermore **Phelan** and associates: Arbitrarily defined AFI 5cm as very low, 5.1 – 8cm as low and >8.1cm or > 20cm as high.

Rhelan JP, Smith CV, and Ruther Ford (1987). Have also found an inverse relationship between AFI and non reactive NST, intrapartum foetus heart rate decelerations, meconium staining, caesarean section, foetal distress and low apgar scores.

The relationship of marginal and decreased amniotic fluid volumes to perinatal outcome:

Manning et al (1979): Have reported an increased perinatal mortality in foetus in whom the largest pocket of amniotic fluid measured <1cm.

Mercer et al (1984): In a survey of pregnancies complicated by increased amniotic fluid had shown various congenital anomalies to be associated with oligohydramnios. Most commonly those involving the genitourinary system, renal agenesis, obstructive uropathy, prune belly syndrome, multicystic dysplastic kidney, thanatophoric dwarfism, agenesis of thyroid gland, heart block and multiple anohiatus. Exposure to ACE inhibitors have been associated with oligohydramnios. Oligohydramnios early in pregnancy is associated with poor foetal outcome of both cause and effect.

Mercer and Brown (1986): Described 34 mid trimester pregnancies complicated by oligohydramnios, diagnosed ultrasonically by absence of amniotic fluid pockets greater than 1cm in any vertical plane.

Nine of these fetuses (26%) had anomalies and 10 of the 25 who were phenotypically normal either aborted spontaneously or were still born because of severe maternal hypertension, retarded foetal growth or placental abruption of 14 live born infants 8 were preterm and of which 7 died. The 6 infants who were delivered at term did well thus there were only 7 surviving infants born to the 34 women with severe early onset oligohydramnios.

Similarly **Schiummer et al** (1991) And **Robson et al** (1992): have demonstrated oligohydramnios to be associated with the diagnosis of foetal distress and with an increased frequency of caesarean delivery.

Quetel and Associates (1992): visualized ultrasonically 13 foetus who had severe oligohydramnios after amnioinfusion with warm saline coloured with indigo carmine. In 11 karyotyping was also obtained and in all the foetus anatomical assessment was possible after amnio infusion. In most cases they were able to make definite diagnosis.

Otherwise normal infants may suffer the consequences of severely diminished amniotic fluid, because adhesion between amnion and foetus parts may cause serious deformities including amputation moreover subjected to pressure from all sides the foetus assumes a peculiar appearance and musculoskeletal deformities such as club foot, are observed frequently typically in cases of oligohydramnios the skin of the foetus appears dry leathery and wrinkled.

Groome and associate (1991): Have shown oligohydramnios and increased foetal urine production prior to labour may also be a marker for infants who may not tolerate labour well.

Grubbs and Paul (1992): Demonstrated significant oligohydramnios defined by an AFI of less than 2cm is also associated with an increased risk of prolonged foetal heart rate deceleration in labour.

Druzin and Adams (1990): Found a positive correlation between meconium and the absence of fluid at the time of amniotomy.

Sarno et al shoed that when the AFI was <5cm the incidence of meconium staining and variable decelerations increased.

According to **Sadovsky et al** (1989): The use of prophylactic amnio infusion in patient with meconium staining seemed to have some positive effects on perinatal outcome.

Stormy et al (1990) found that prophylactic amnio infusion in patient with an initial AFI of <5cm reducing the frequency of variable deceleration, meconium passage and operative deliveries for foetal distress.

Tong Song T. Srisomboon J – 1993 found that amniotic fluid volume assessment was an effective method in predicting foetal distress in post term pregnancy as compared with the conventional non stress test.

Chauhan sp et al (1998) found a positive effect of prophylactic transabdominal amnioinfusion on the latency period in patients with preterm premature rupture of membranes and oligohydramnios. Improvement in foetal heart rate, short term variability associated with progressive increase in amniotic fluid volume, as an expression of foetal well being.

Placenta and Placental Grading:

The normal anatomy of the placenta and the retroplacental area can be well defined with gray scale sonography (Spirt BA, Kagan EH, Rozanski RM 1979). The placenta has a characteristic granular echo pattern, with strong echoes emanating from the chorionic plate. After 37 weeks, strong echoes representing calcification may appear; this is a normal physiologic process. The significance of early calcification is unclear. The retroplacental myometrium and venous drainage of the placenta are easily visualized by sonography. Variations in the appearance of the retroplacental myometrium result from different degrees of bladder filling and from uterine contractions. These normal retroplacental structures should not be mistaken for myomas or haematomas.

Toshio Fujikura (1963): Said that calcification in the placenta has been considered as a sign of the maturity because it is found frequently in variable degrees in full term placentas. The deposits appear on or near the maternal decidual surface and are usually demonstrable in gross examination. It is only when the calcium deposits are excessive that terminal or stem villi and chorion are affected equally. No correlation between the amount of calcium in the placenta and the health of the mother has been described.

The study was carried out at university of Oregon medical school hospitals and clinics on 1048 term placentas of live and stillborn infants. Of the 1048 placentas 150 (14.3%) contained moderate and marked degrees of calcium deposits in either the decidual basalis, terminal or stem villi, chorion or a combination of these. The incidence of placental calcification seemed to decrease with advancing maternal age The average weights of the infants and the placentas reveal similar values in the non-calcified and calcified groups.

C.C. Fisher , William Garett, George Kossoff (1975): Conducted a study and found that with gray scale ultrasonic echography, it is possible to identify changes in the placental anatomy which formerly have been recognized only by examination of the placenta after delivery. They also noted an association between the premature appearance of aging of the placenta and a decline in placental function. They found that the ultrasonic characteristics of placenta which occur in late pregnancy if occurred at an earlier stage in pregnancy would result in small for date baby at delivery.

The association of premature appearance of grade III changes in the placenta in pregnancies complicated by maternal diseases known to produce a decline in placental function, suggest that incidental finding of grade III placental changes at the time of ultrasound may indicate unrecognized pregnancy complications.

Grannum et al (1979): Reported premature appearance of grade III placental changes in pregnancies complicated with Intra Uterine Growth Retardation (IUGR).

Montan et al (1980): Observed no correlation between the mean birth weight and different placental grades and reported that grade III placenta was not a predictor of adverse foetal outcome.

Ruth A. Petrucha, Lawrence D. Platt (1981): Conducted a study to show the relationship of placental grade to gestational age. The placenta was graded according to the system described by Grannum and associates. They graded the placenta from 0 to III based on changes in the (i) Chorionic plate; (ii) Placental substance and (iii) Basal layer.

Progressive changes in ultrasonic placental architecture are a function of increasing gestational age. This relates to recent speculation that the grade III placenta may be a marker for pulmonary lung maturity. 95% of grade III placentas appear beyond 35 weeks of gestation. This corresponds quite closely to the gestational age at which the abrupt rise in mature L/S ratio occurs. It was found that in pregnancies in which grade III placenta appeared in <35 weeks of gestation – Intrauterine growth retardation was found in the fetus.

Kamla Ganesh, UMA Dhawan and N.C. Gupta (1982): They carried out their study on 100 primigravidae, having gestation beyond 30 weeks with known LMP and BP > 140/90mm Hg (study group) and 40 normotensive women (control group). They were studied for placental grading by ultrasound and the findings were correlated with fetal outcome. Grade I placenta was significantly less common in the study group as compared to controls ($P<0.01$). Grade III placenta was significantly more in patients of severe PIH as compared to mild PIH ($P<0.001$). preterm babies were born when placenta was grade II and III in 12.5% and 18.8% of the cases respectively, As compared to none in the normotensive controls, indicating accelerated placental maturity in PIH. IUGR babies resulted in 7.1% of the cases of PIH with grade I and 23.2% with grade II and 31.1% with grade III placenta.

R. William Quinlan, Amelia C. Cruz, William C. Buhi, Magdeline Martin (1982): Changes in placental texture and structure on ultrasonic examination have been suggested as correlating to advancing fetal pulmonary maturity. The association of preterm appearance of grade III changes in the placenta in the complicated pregnancy suggests that these changes are associated with a decrease in placental function and premature senescence of the placenta. They found a high incidence of perinatal problems in association with preterm appearance of grade III changes. Perinatal complications

included maternal hypertensive disorders, Intrauterine growth retardation, abruptioplacentae, and fetal distress in labour. According to them, the high incidence of these disorders in preterm pregnancies with grade III changes in the placenta suggest that the sonographic changes found reflect placental dysfunction or senescence rather than normal maturational development. The appearance of grade III changes in the placenta in the preterm pregnancy is suggested as a predictive indicator of potential perinatal problems in late pregnancy.

Woods DL, Malan AF, Heese HD (1982): In their study they compared the placental size between appropriate for gestational age (AGA) and small for gestational age (SGA) infants born at term. They found that the placental weight, chorionic plate area and villous surface area were significantly reduced in the small for gestational age infants although the ratio of placental weight to the birth weight was similar in the AGA and SGA infants, the latter had significantly under weight placentas for their head circumference and crown heel length. The ratios of placental weight to assess brain weight and villous surface area to assess brain weight were also significantly reduced in the SGA infants. They concluded that SGA infants had both absolutely and relatively small placentas.

Prinz W, Schuhmann RA, Kalbfleisch W (1983): They investigated morphologically the placentas of 105 SFD infants and 190 newborns of normal birth weight. Each of these two groups consisted of premature newborns and mature newborns. Macroscopic and microscopic data were evaluated by means of a so called data bank. The weight and the area of attachment were determined. The hisotlogic findings were classified in two groups:

(1) Disturbances of placental maturation.

(2) Morphologic changes in consequence of reduced uteroplacental blood flow.

A marked increase of hypoplastic placentas was found among immature and mature small for date infants. Retarded placental maturity was found more frequently among mature small for date infants than among prematures. Among these, placentas with signs of a reduced uteroplacental perfusion were found more frequently.

Kazzi GM, Thomas Gross, Robert J. Sokol, Nadya J. Kazzi (1983): Advanced placental maturity (grade III), as determined on ultrasound examination, has previously been reported to be a marker of term gestation. In their study of 109 pregnancies which resulted in the birth of infants weighing less than or equal to 2700gm, The hypothesis that a grade III placenta, according to Grannum's classification, can differentiate small for gestational age (SGA) infants from small non-SGA infants was tested. Of the study patients, 44 had grade III placentas and 65 had non-grade III (O, I, II) placentas with in week of delivery. The presence of grade III placenta was followed by the delivery of a SGA infant 59% of the time, and 62% of the SGA infants could be correctly identified ($P<0.001$). The association of a grade III placenta and SGA birth was maintained in patients at less than or equal to 34 weeks of gestation. They found in their study that grade III placenta was significantly related to the delivery of SGA infants with a true positive rate of 62% and a sensitivity of 66% (P less than 0.008) these results were consistent with the concept that for small fetuses documentation of "Maturity" can be used to discriminate those with IUGR from those without this problem. Further more, placental "Maturation", as detected sonographically, appears to be accelerated in association with IUGR, consistent with the anatomic concept of premature placental senescence. They

concluded that, in situations in which the fetus is known to be small, sonographic grading of the placenta may be helpful in detecting IUGR.

Robert M. Patterson, Robert H. Hayashi and Dora Cavazos (1983): Perinatal outcome in 398 patients who had grade II or grade III placentas was analyzed in a cross sectional study. Among the 398 study patients, a grade II placenta was observed in 221, and a grade III placenta was observed in 177. The mean gestational age at which a grade II placenta was observed was 33.1 ± 1.9 weeks. Grade III placentas were observed at a mean gestational age of 36.3 ± 2.2 weeks. Early placental maturation was identified in 21 patients in the grade II population and in 30 patients in the grade III population. A trend toward lower mean birth weights was identified in the group with early placental maturation as compared to controls, however statistical significance was achieved only in the grade II population. The placental maturation was not significantly affected by maternal age or parity. The significant difference in mean birth weight between patients with early placental maturation and matched control patients in the grade II population was not present in the grade III population. The incidence of SGA infants in patients with early placental maturation versus control patients in the grade III population was significantly increased. The sensitivity of early placental maturation as a marker for SGA infants was 46%, with a predictive value of 16.7%.

Khalil MA Tabsh (1983): Reported that preterm appearance of grade III placenta was associated with adverse perinatal outcome.

Patterson et al (1983): Did not find any correlation of early placental maturation with poor perinatal outcome. He observed that sensitivity of early placental maturation as a marker of small for gestational age infant was 46% with a predictive value of 16.7%.

Hopper KD, Komppa GH, Bicep, Williams MD, Cotterill RW, Ghaed N, (1984): Conducted a study and found that pregnancies complicated by pre-eclampsia or IUGR have an earlier and faster placental maturation than normal.

Kopernik H, Schwarz B. (1985): Conducted a study in 784 pregnant women to show the effect of early sonographic placental maturity on birth weight. They found that early placental maturity resulted in children with low birth weight. In children with birth weight over 3500gm first findings of placental maturity were in 81.8% after 35 weeks gestation. According to them, there may be possible correlations between placental maturity and risk factors typical for low placental perfusion with the consequence of FGR as hypertension, hypotension, stress and smoking.

Luckert G., Loffler F., Kamin G., Domken (1986): Influence of sonographically demonstrable changes of placental structure on prematurity and intrauterine retardation was analysed and concerning its statistical rehability in 807 gravids. While the stages 0 and 1 don't effect prematurity and intrauterine retardation, the stage 2 is accompanied. Premature infant of normal birth weight if proved before the 32nd week of pregnancy if stage 3 appears before the 34 week of pregnancy we found in 64% a normal weight and in 80% a small for date premature newborn. In comparison with the normal weight newborn the difference is statistically significant with an error probability of alpha = 0.05.

Miller JM Jr, Brown HL, Kissling GA, Gabert HA (1988): They carried out their study on 246 term patients. Among 246 term patients undergoing ultrasonic evaluation with in 1 week of delivery, a grade III

placenta was found in 39.4%. They found that at term, advanced placental maturity is not associated with aberrant fetal growth.

Suska P, Vierik J, Handzo I, Krizkom (1989): They carried out a study and found that macroscopic examination of the placenta in newborns with intrauterine growth retardation reveals changes responsible for reduced placental functions. These can then be diagnosed in utero, e.g. by ultrasonic examination. The most serious defects include reduced weight an insertion area of the placenta and infarcts covering more than 6% of the placental area. Extrac chorial placentas also have adverse affects.

Chitlange SM, Hazari KT, Joshi JV, Shah RK, Mehta AC (1990): Reported that preterm grade III placenta is found to be a sensitive predictor of poor perinatal outcome.

Salafia CM, Vintzileos AM, Silberman L, Bantham KF, Vogelca (1992): Decreased birth weight was associated with placental infarction.

Veena Agrawal, Sapna Jain (2000): Reported that premature aging of placenta was an indication of decline in its function and was found to be associated with an increased incidence of maternal and foetal complications in the form of hypertension, IUGR, fetal demise abruptioplacentae and Perinatal mortality. Presence of grade III placenta requires close monitoring with the continuation of pregnancy.

Sanjay Kumari, Harjeet Sawhney, Kala Vasishta, Anil Narang (2001): They carried out a study in 50 pregnant women with IUGR and in 50 pregnancies with fetal growth appropriate for gestational age. Ultrasonic assessment of placental grading was done in these patients. The incidence of

grade III placentae and its relation with perinatal outcome was assessed in both the groups. In growth retarded fetuses, incidence of grade III placenta was 58% and it was 36% in fetuses appropriate for gestational age. Birth weight was significantly lower in grade III placenta (1482.3 ± 320.5g) compared to grade II placenta (1766 ± 484.7gm) in growth retarded fetus. Grade III placenta was associated with higher incidence of perinatal death. In the control group, placental grading had no correlation with perinatal outcome.

Materials & Methods

MATERIAL AND METHODS

The present study was conducted in the department of obstetrics and gynaecology, M.L.B. Medical College, Jhansi. The cases were selected from patients attending antenatal clinic and labour room in the department of obstetrics and gynaecology. The study was done over a 12 month period from Feb 2004 – Feb 2005.

Criteria for Selection of cases:

Following criterias were used for selection of cases in both the groups:

1) **Period of Gestation:** Patients included were with 35 or more weeks of pregnancy.
2) **Age:** Patients with age group between 18 – 35 years were included.
3) **Parity:** Both primigravida and mutligravida were selected.
4) Singleton pregnancy with known gestational age and delivery with in 7 days of last ultrasound examination.
5) **Presentation:** All patients irrespective of the presentation were included.
6) **Status of membranes:** Cases only with intact membranes were included.

Exclusion Criteria:

Patients with premature rupture of membranes were excluded from the study.

Study Subjects:

A total of 160 cases were included in our study. The cases were divided in two groups:

Control Group: This group included 80 antenatal cases with 35 weeks pregnancy or more with fetal growth appropriate for gestational age and without any antenatal complications in the mother.

Study Group: This group included 80 antenatal cases with 35 weeks pregnancy or more with diagnosis of intrauterine growth retardation and with 40% of the women having antenatal complications.

Each selected patient was subjected to a detail history, examination and investigations.

History: A detailed history of each selected case was taken in the following lines:

General Particulars:

Name

Age

Gravidity

Socio economic status

Education

Presenting complaints:

Detailed history regarding period of amenorrhoea.

Date of quickening

Swelling over feet or body, headache blurring of vision

Epigastric pain

Premature contractions or leaking per vaginum.

Obstetric history:

Obstetric history was taken in detail regarding

- Parity
- Number of abortions
- Previous still birth
- Delivery of growth retarded baby
  Or perinatal death

Menstrual History:

The age of menarche, regularity of cycles was noted and a careful history of last menstrual period was asked for.

Personal History:

Socioeconomic status of the patient was estimated by inquiring about the income of the family and the number of family members.
History of any addiction of tobacco or alcohol was also enquired.

Past History:

History of chronic hypertension, tuberculosis and diabetes mellitus was taken.

Examination:

Examination was done under the following headings:

1) General Examination:

General condition of the patient
Pulse rate
Blood pressure
Temperature
Pedal edema
Dehydration
Pallor or icterus were noted

2) Systemic Examination:

This included examination of cardiovascular, respiratory and nervous system.

Obstetric Examination:

The per abdominal examination was done to ascertain fundal height, lie, presentation and position of the presenting part; foetal heart sound rate, Rhythm, Variation with contraction amount of liquor amnii; uterine contractions.

Per speculum Examination:

Per speculum examination was done to rule out any vulval, vaginal or cervical infection and also to see leaking per vaginum.

Pervaginal Examination:

Pervaginal examination was done to assess dilatation of cervix, effacement and consistency of cervix and status of membranes. Station of head and a complete assessment of the pelvis was done to rule out cephalopelvic disproportion.

Investigations:

Routine Investigations:

Haemoglobin

Total and differential leucocyte count

Blood group: ABO Rh

Urine examination Albumin, Sugar and microscopic examination.

USG Examination:

Included the examination of the foetus, the placenta and the liquor.

The foetus was examined under the following headings:

Foetus: Single / Twin (only singleton pregnancy included)

Presentation: Cephalic / Breech / Oblique.

Cardiac and somatic activity was assessed.

Heart rate calculated in beats/Minutes.

Placenta: Its site and grading done.

Amniotic fluid seen whether less / normal

Amniotic fluid index calculated, sonographic estimation of foetal weight done.

Placenta was graded according to the system described by **Grannum et al** (1979) and amniotic fluid index was estimated by four quadrant technique as described by **Phalen et al** (1987).

Technique:

Examination is performed with patient in supine position. Using landmarks on the maternal abdomen the uterine cavity is divided into four quadrants. The umbilicus marks the division between the upper and the lower halves and the linea nigra divides it into right and left halves.

For all measurements the linear transducer head is placed along the mothers longitudinal axis and held perpendicular to the floor in the saggital plane. The maximum vertical dimension of the largest pocket is measured in centimeters. Vertical is defined as perpendicular to the transducer head.

The measurements obtained from each quadrant were summed to form the amniotic fluid index.

The pregnancy outcome was noted in the form of:

- Occurrence of fetal distress (Abnormal heartrate, meconium staining of liquor).
- Mode of delivery vaginal / abdominal.
- Fetal weight / sex.
- Apgar score at 1 minute and 5 minutes.
- Need for endotracheal intubation of baby.
- Admission to intensive care unit.
- Total duration of hospital stay and neonatal mortality.

Observations

OBSERVATIONS

A total of 160 patients presenting with normal and abnormal (Complicated with IUGR) pregnancy beyond 35 weeks of gestation were included in our study. The cases were divided into two groups:

Control Group (80 cases):

Included pregnant women with fetal growth appropriate for gestational age.

Study Group (80 cases):

Included pregnant women with IUGR. Diagnosis of IUGR was made by clinical examination and on ultrasonography when estimated fetal weight was <2SD of the mean weight at a particular gestation.

Following observations were made in our study.

Table – 1

Distribution of cases

Group	No. of cases	
Study group	80	Included pregnant women with IUGR.
Control group	80	Included pregnant women with fetal growth appropriate for gestational age.

Table – 2

Age distribution

Age in years	Study group		Control group	
	No.	%	No.	%
≤20	11	13.75	9	11.25
21 – 25	58	72.50	60	75.00
26 – 30	7	8.75	8	10.00
31 – 35	4	5.00	3	3.75
Total	**80**	**100**	**80**	**100**
Mean ± SD	**23.46 ± 2.91**		**23.48 ± 2.73**	

Table – 2 shows that maximum cases (73.75%) in both the groups were young between 21 – 25 years of age group. Both the study and control groups were comparable regarding the age of patients. The difference was not statistically significant (P value >0.05).

Table – 3

Gravidity

Gravidity	Study group		Control group	
	No.	%	No.	%
Primigravida	43	53.75	72	90
Multigravida	37	46.25	8	10
Total	**80**	**100**	**80**	**100**

In the present study both primigravida and multigravida were included. Majority of the women in control group were primigravida (90%) and in the study group 37 (46.25%) were multigravida and 43 (53.75%) were primigravida.

Table – 4

Maternal weight distribution

Weight (In kg)	Study group		Control group	
	No.	%	No.	%
≤ 50	10	12.5	8	10.0
51 – 55	36	45.0	30	37.5
56 – 60	30	37.5	36	45.0
61 – 65	4	5.0	6	7.5
Total	**80**	**100**	**80**	**100**
Mean ± SD	**54.88 ± 3.51**		**55.60 ± 3.67**	

Table – 4 shows weight distribution of women in study and control groups. Majority of the women in study group had weight less than 56kg (46; 57.5%). 30 (37.50%) had weight between 56 – 60kg and 4 (5%) had weight between 61 – 65kg whereas in control group, majority of the women had weight >55kg (42, 52%); 38 (47.5%) had weight less than 56kg, and 6 (7.5%) had weight between 61 – 65kg. But the difference was not statistically significant.

Table – 5

Residential area

Residential Area	Study group		Control group	
	No.	%	No.	%
Urban	25	31.25	30	37.50
Rural	55	68.75	50	62.50
Total	**80**	**100**	**80**	**100**

More than half of patient's in both the groups were from rural background. In study group 55 (68.75%) patients were from rural background and in control group 50 (62.5%) patients were from rural background.

Table – 6

Educational Level

Educational Level	Study group		Control group	
	No.	%	No.	%
Illiterate	30	37.50	18	12.50
Primary	20	25.00	26	32.50
High School	14	17.50	18	22.50
Intermediate	10	12.50	12	15.00
University	6	7.50	6	7.50
Total	**80**	**100**	**80**	**100**

Table – 6 shows that 30 (37.5%) patients in study group were illiterate, where as in control group 8 (10%) patients were illiterate.

Table – 7

Socioeconomic Status

Social Class	Study group		Control group	
	No.	%	No.	%
I	1	1.25	3	3.75
II	2	2.50	5	6.25
III	10	12.50	36	45.00
IV	42	52.50	28	35.00
V	25	31.25	8	10.00
Total	**80**	**100**	**80**	**100**

The patients were divided into 5 social classes according to modified BG Prasad classification for 1994. According to this classification social class I, II, III, IV and V included the patients with per capita income of >1400 Rs. Per month, 700 – 1399, 420 – 699, 211 – 419 and below 210 Rs. Per month respectively.

In the study group majority of the patients (42; 52.50%) were in class IV and minimum number of patients (1) were in class I. In control group, majority of the patients (36; 45%) were in class III and minimum were in class I. Class IV included (28; 35%) patients.

Table – 8

Antenatal Complications

Antenatal Complications	Study group		Control group	
	No.	%	No.	%
PIH	28	35.00	-	-
Heart disease	2	2.50	-	-
Toxoplasmosis	2	2.50	-	-
Total	**32**	**40.00**	-	-

In the study group 32 (40%) women had antenatal complications in the present pregnancy. While in control group all pregnancies were uncomplicated.

Table – 9

Placental grading in study and control groups

Group	Grade II		Grade III	
	No.	%	No.	%
Study group	34	42.50	46	57.50
Control group	51	63.75	29	36.25

In the study group 34 (42.5%) patients had grade II placental changes and 46 (57.5%) patients had grade III placental changes. Whereas in control group 51 (63.75%) patients had grade II placental changes and 29 (36.25%) patients had grade III placental changes. Incidence of grade III placenta was high in the study group as compared to control group.

Table – 10

Placental grading in control group in relation to gestational age

Gestational age (in weeks)	Grade II		Grade III	
	No.	%	No.	%
35 – 36	1	1.96	1	3.46
36.1 – 37	5	9.80	3	10.34
37.1 – 38	22	43.14	10	34.48
38.1 – 39	15	29.41	10	34.48
39.1 – 40	8	15.69	5	17.24
Total	**51**	**100**	**29**	**100**
Mean ± SD	**38.03 ± 0.3**		**38.07 ± 0.83**	

Table – 10 shows that there were 22 cases with 37.1 – 38 weeks of gestation who had grade II placental changes and 10 cases had grade III placental changes.

In 35 – 36 weeks gestation, 1 case had grade II placental changes and 1 case had grade III placental changes.

In 36.1 – 37 weeks gestation, 5 cases had grade II placental changes and 3 cases had grade III placental changes.

In 38.1 – 39 weeks gestation, 15 cases had grade II placental changes, and 10 cases had grade III placental changes.

In 39.1 – 40 weeks gestation, 8 cases had grade II placental changes and 5 cases had grade III placental changes.

Table – 11

Placental grading in study group in relation to gestational age

Gestational age (in weeks)	Grade II		Grade III	
	No.	%	No.	%
35 – 36	13	38.24	17	36.96
36.1 – 37	12	35.29	19	41.30
37.1 – 38	7	20.59	8	17.39
38.1 – 39	1	2.94	1	2.17
39.1 – 40	1	2.94	1	2.17
Total	**34**	**100**	**46**	**100**
Mean ± SD	**36.50 ± 1.05**		**36.44 ± 1.09**	

Table – 11 shows that in 35 – 36 weeks gestation, there were 13 cases who had grade II changes and 17 cases had grade III changes.

In 36.1 – 37 weeks gestation, 12 cases had grade II changes and 19 cases had grade III changes.

In 37.1 – 38 weeks gestation, 7 cases had grade II placental changes, and 8 cases had grade III placental changes.

In 38.1 – 39 weeks gestation, 1 case had grade II placental changes and 1 cases had grade III placental change.

In 39.1 – 40 weeks gestation, 1 case had grade II and 1 case had grade III placental changes.

Table – 12

Amniotic fluid index in study and control groups

Amniotic fluid index (in cm)	Study group		Control group	
	No.	%	No.	%
≤ 5	20	25.00	1	1.25
5.1 – 8	47	58.75	3	3.75
8.1 – 11	10	12.50	8	10.00
11.1 – 14	3	3.75	49	61.25
14.1 – 17	0	0.00	19	23.75
17.1 – 20	0	0.00	0	0.00
Total	**80**	**100**	**80**	**100**
Mean ± SD	**6.14 ± 2.53**		**12.61 ± 2.39**	

Statistical calculations were done in this table and P value was < 0.01 signifying that there is a highly significant statistical difference in the AFI values in the control and study group.

Amniotic fluid index was calculated in all patients in both the groups.

In the control group, 68 patients (85%) had AFI ranging from 11.1 – 17cm. 3 patient (3.75%) had AFI ranging from 5.1 – 8cm; and 8 patients (10%) had AFI ranging from 8.1 – 11cm; and 1(1.25%) patient had AFI in the range of 0 – 5cm.

In study group, 47 patients (58.75%) had AFI ranging from 5.1 – 8cm, 20 patients (25%) had AFI ranging from 0 – 5cm; 10 patients (12.5%) had AFI ranging from 8.1 – 11cm, and 3 patients (3.75%) had AFI ranging from 11.1 – 14cm.

Table – 13

Amniotic fluid index in control group in relation to gestational age

Gestational age (in weeks)	No. of cases	Amniotic fluid index (Mean)
35 – 36	2	15.5
36.1 – 37	8	13.8
37.1 – 38	32	13.6
38.1 – 39	25	12.6
39.1 – 40	13	11.3

AFI when calculated in relation with gestational age in control group was as follows:

There were 2 cases with 35 – 36 weeks gestation who had mean AFI = 15.5.

There were 9 patients with 36.1 – 37 weeks gestation with mean AFI = 13.8.

27 patients with 37.1 – 38 weeks gestation had mean AFI = 13.6.

29 patients with 38.1 – 39 weeks gestation had mean AFI = 12.6 and 13 patients with 39.1 – 40 weeks gestation had mean AFI = 11.3.

Table – 14

Amniotic fluid index in study group in relation to gestational age

Gestational age (In weeks)	No. of cases	Amniotic fluid index (Mean)
35 – 36	30	6.7
36.1 – 37	31	7.1
37.1 – 38	15	8.4
38.1 – 39	2	4.5
39.1 – 40	2	5

Amniotic fluid index in relation with gestational age in study group was as follows:

There were 30 patients with 35 – 36 weeks gestation who had mean AFI = 6.7. There were 31 patients with 36.1 – 37 weeks gestation who had mean AFI = 7.1. There were 15 patients with 37.1 – 38 weeks gestation who had mean AFI = 8.4. There were 2 patients with 38.1 – 39 weeks gestation who had mean AFI = 4.5. There were 2 patients with 39.1 – 40 weeks gestation who had mean AFI = 5.

Table – 15

Placental grade and fetal distress in control group

Fetal distress	Grade II		Grade III	
	No.	%	No.	%
Present	3	5.88	2	6.90
FHR Abnormality	2	-	2	-
Meconium	1	-	-	-

Fetal distress was present in 3 (5.88%) patients in grade II placenta and in 2 patients (6.90%) in grade III placenta in control group. The incidence of fetal distress in labour was similar in grade II and grade III placenta.

Table – 16

Placental grade and fetal distress in study group

Fetal distress	Grade II		Grade III	
	No.	%	No.	%
Present	22	64.71	31	67.39
FHR Abnormality	16	-	19	-
Meconium	6	-	12	-

Fetal distress was present in 22 patients (64.71%) in grade II placenta and 31 patients (67.39%) in grade III placenta in study group. The incidence of fetal distress was similar in grade II and grade III placenta.

The incidence of fetal distress was more in study group than in control group.

Table – 17

Mode of delivery

Mode of delivery	Study group		Control group	
	No.	%	No.	%
Vaginal delivery	50	62.5	76	95
Caesarean section	30	37.5	4	5
Total	**80**	**100**	**80**	**100**

In the control group normal vaginal delivery occurred in 95% patients (76) and caesarean section was done in 5% (4) of patients.

In the study group normal vaginal delivery occurred in 62.5% (50) patients and caesarean section was done in 37.5% (30) patients.

Table – 18

APGAR score at 1 minute

Apgar score	Control group		Study group	
	No.	%	No.	%
0 – 3	0	0	8	10
4 – 6	3	3.75	12	15
7 – 10	77	96.25	60	75
Total	**80**	**100**	**80**	**100**
Mean ± SD	**8.369 ± 0.675**		**7.275 ± 2.28**	

The results of 1 minute apgar scoring was:

In the control group, the apgar score at 1 minute was between 4 – 6 in 3 (3.75%) babies and in 77 (96.25%) babies it was between 7 – 10. In the study group, the apgar score was between 0 – 3 in 8 (10%) babies; between 4 – 6 in 12 (15%) babies and between 7 – 10 in 60 (75%) babies.

Table – 19

Apgar score at 5 minutes

Apgar score at 5 minute	Control group		Study group	
	No.	%	No.	%
0 – 3	0	0	7	8.75
4 – 6	0	0	3	3.75
7 – 10	80	100	70	87.50
Total	**80**	**100**	**80**	**100**

Apgar scoring was also done in both the groups at 5 minute and none of the newborns in control group had low apgar scores. In the study group 7 (8.75%) newborns had apgar score between 4 – 6 and 70 (87.5%) newborns had apgar score between 7 – 10.

Table – 20

Association of placental grade and birth weight in control group

Birth Weight (in kg)	Grade II		Grade III	
	No.	%	No.	%
2.5 – 2.7	8	15.69	9	31.03
2.8 – 3.0	30	58.82	12	41.38
3.1 – 3.3	13	25.49	8	27.59
Total	**51**	**100**	**29**	**100**
Mean ± SD	**2.929 ± 0.20**		**2.889 ± 0.24**	
P Value > 0.05				

Table 20 shows association of placental grading with birth weight in control group. In patients with grade II placental changes the mean birth weight was 2.929 and with grade III placental changes the mean birth weight was 2.889 kg which was slightly less than grade II. However, this difference was not statistically significant. (P>0.05).

Table – 21

Association of placental grade and birth weight in study group

Birth weight (in kg)	Grade II		Grade III	
	No.	%	No.	%
1.4 – 1.6	1	2.94	9	19.57
1.7 – 1.9	7	20.59	16	34.78
2.00 – 2.2	17	50.00	21	45.65
2.3 – 2.5	9	26.47	-	-
Total	**34**	**100**	**46**	**100**
Mean ± SD	**2.056 ± 0.47**		**1.878 ± 0.22**	
P Value < 0.05				

The mean birth weight in patients with grade III placental change was 1.878 ± 0.22kg which was significantly lower as compared to grade II placenta. (P <0.05).

Table – 22

Perinatal outcome in study and control

Parameters	Study group		Control group	
	No.	%	No.	%
Live born	76	95%	80	100
Still born	4	5%	-	-
Neonatal death	9	11.25%	-	-
NICU admission	21	26.25%	2	2.5%
Perinatal death	13	16.25%	-	-

Perinatal mortality 162.5 per 1000 live birth

In study group, 76 babies (95%) were live born, 4 (5%) babies were still born 9 babies (11.25%) died in the neonatal period. There was no such adverse perinatal complications found in control group. 21 babies out of 80 in study group were admitted in NICU where as in control group only 2 babies were admitted.

There were 13 perinatal deaths in study group and none in control group. The perinatal mortality in study group was 162.5 per 1000 live birth.

Table – 23

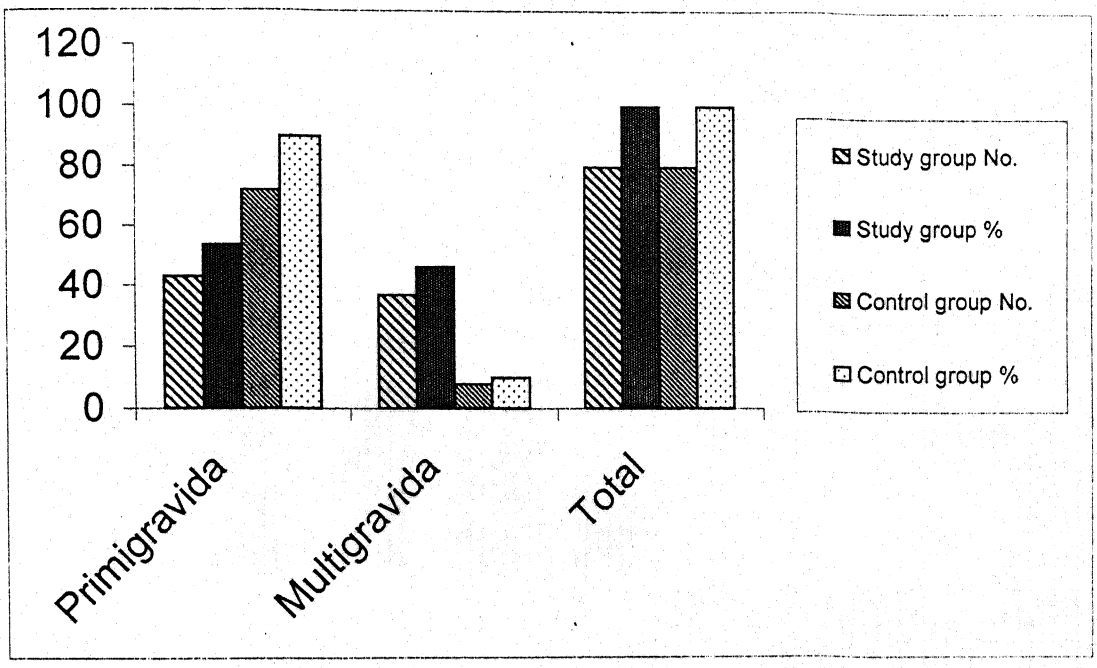
Perinatal outcome in study group in relation to placental grade

Parameters	Grade II		Grade III	
	No.	%	No.	%
Live born	33	97.06	43	93.48
Still born	1	2.94	3	6.52
Neonatal death	2	5.88	7	15.22
NICU admission	4	11.76	17	36.96
Perinatal death	3	8.82	10	21.74
Perinatal mortality	88.23 Per 1000 live birth		217.39 per 1000 live birth	

In study group, in grade II placenta, 33 babies (97.06%) were live born and 1 baby (2.94%) was still born. 3 babies (8.82%) died in the neonatal period, 4 babies (11.76%) were admitted to NICU.

In grade III placenta, 43 babies (93.48%) were live born and 3 babies (6.52%) were stillborn 7 babies (15.22%) died in the neonatal period and 17 babies (36.96%) were admitted in NICU.

GRAVIDITY


120
100
80
60
40
20
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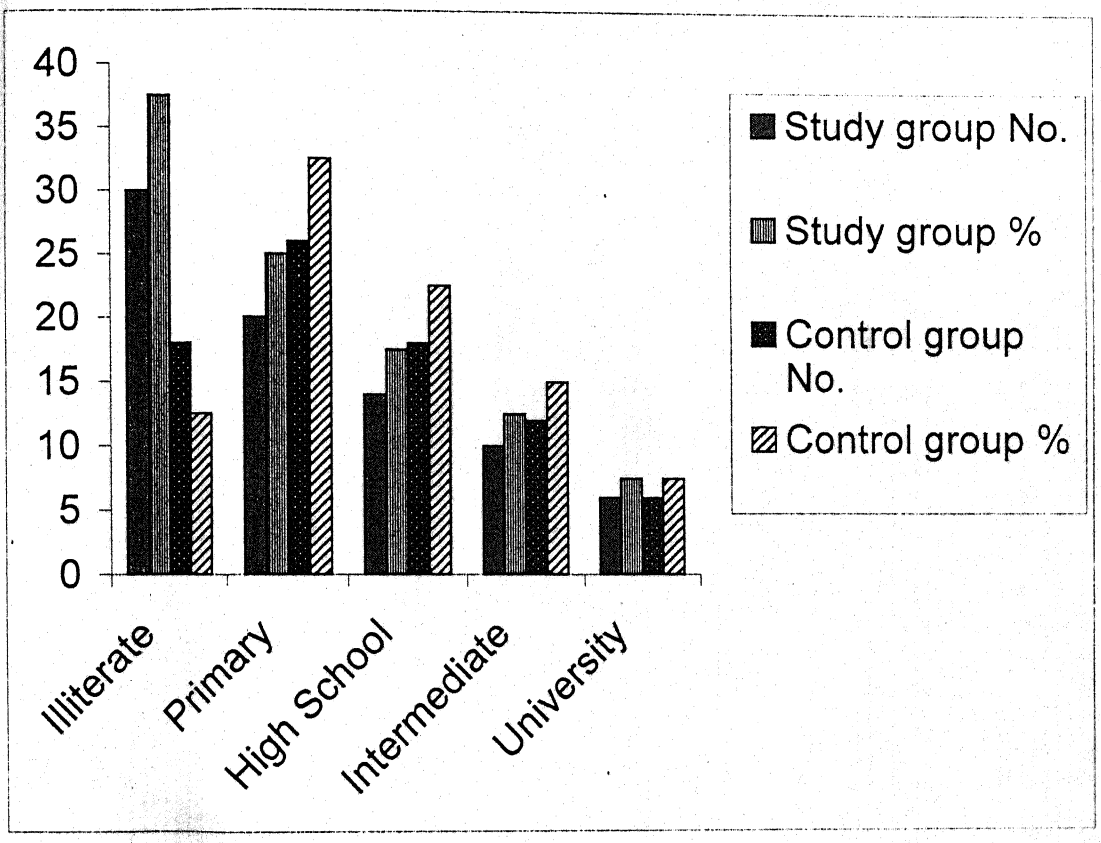
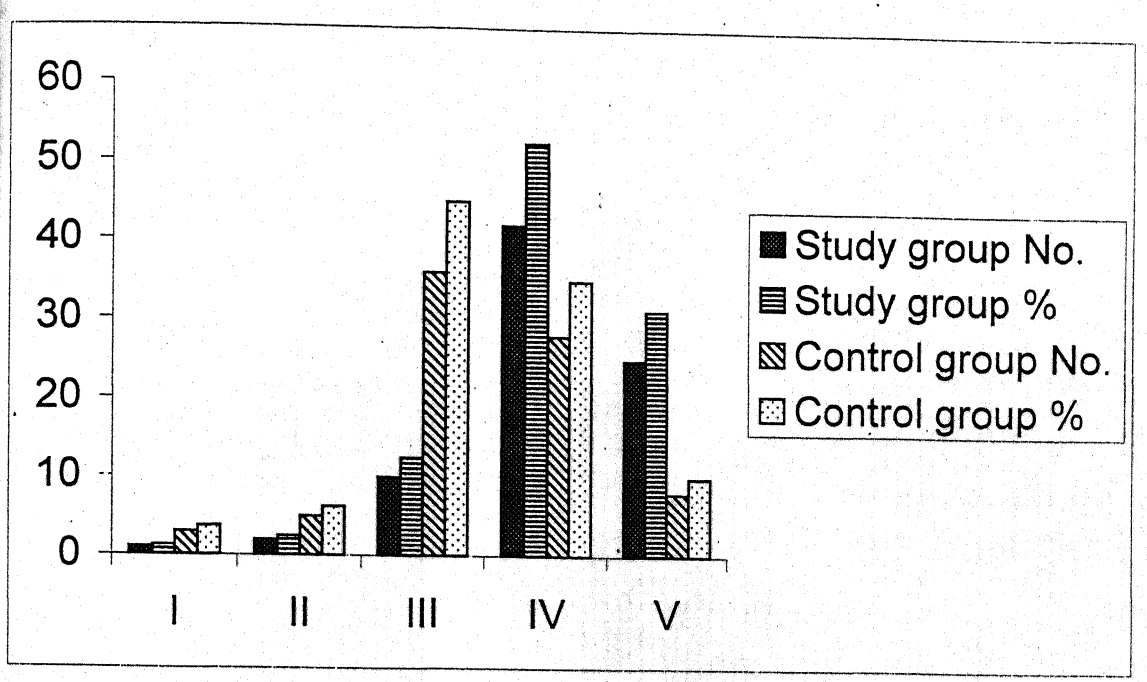
Primigravida
Multigravida
Total
Study group No.
Study group %
Control group No.
Control group %

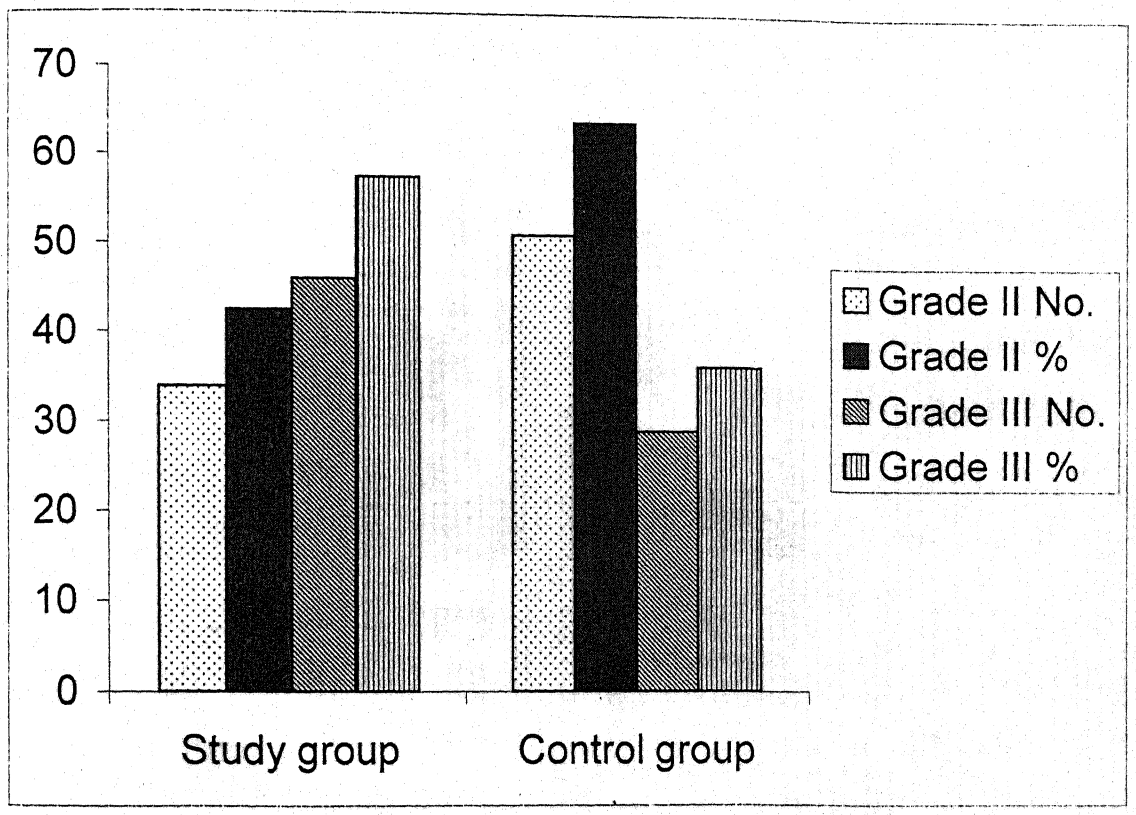
Educational level



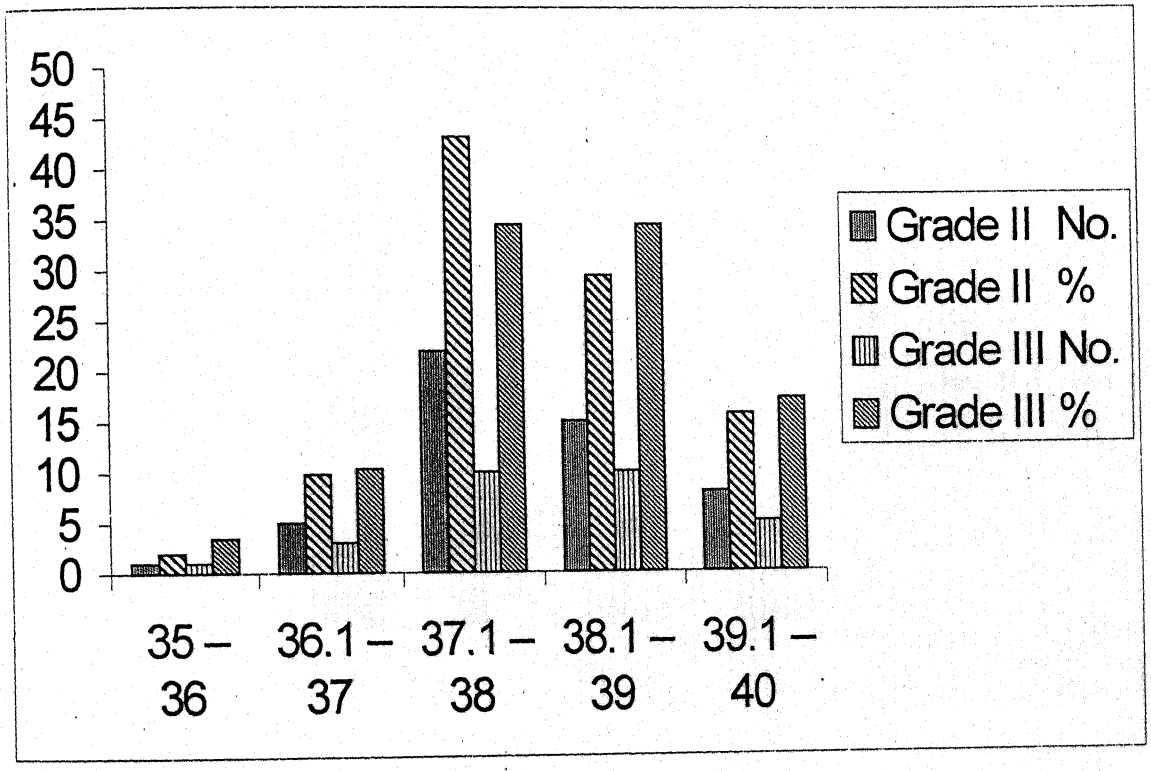
Socioeconomic Status



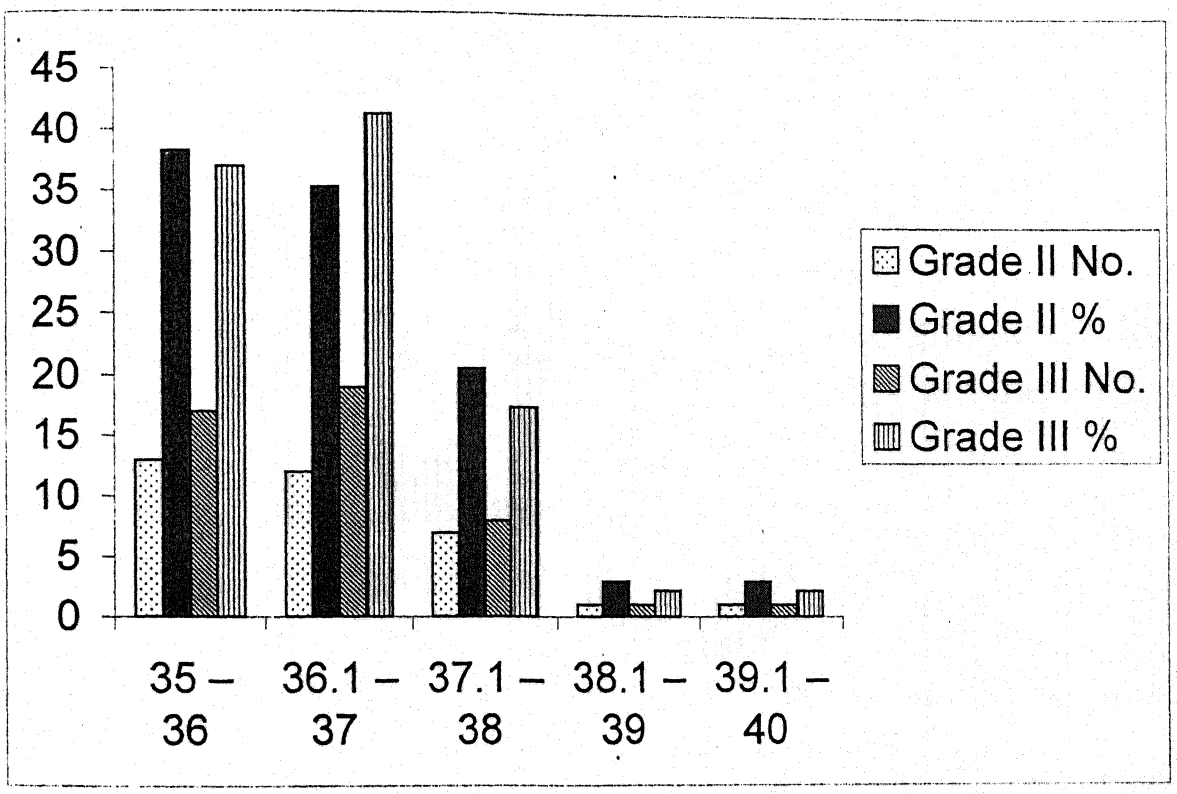
Placental Grading In Study and Control Groups



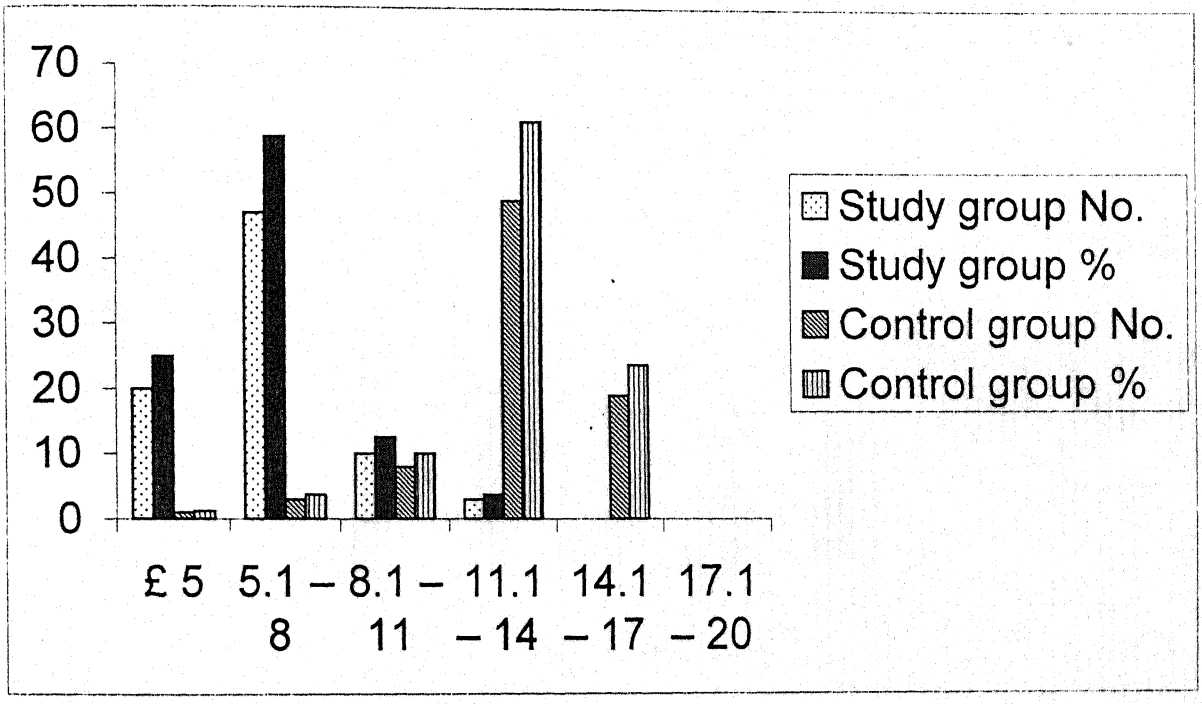
Placental Grading In Control Group in Relation to Gestational Age



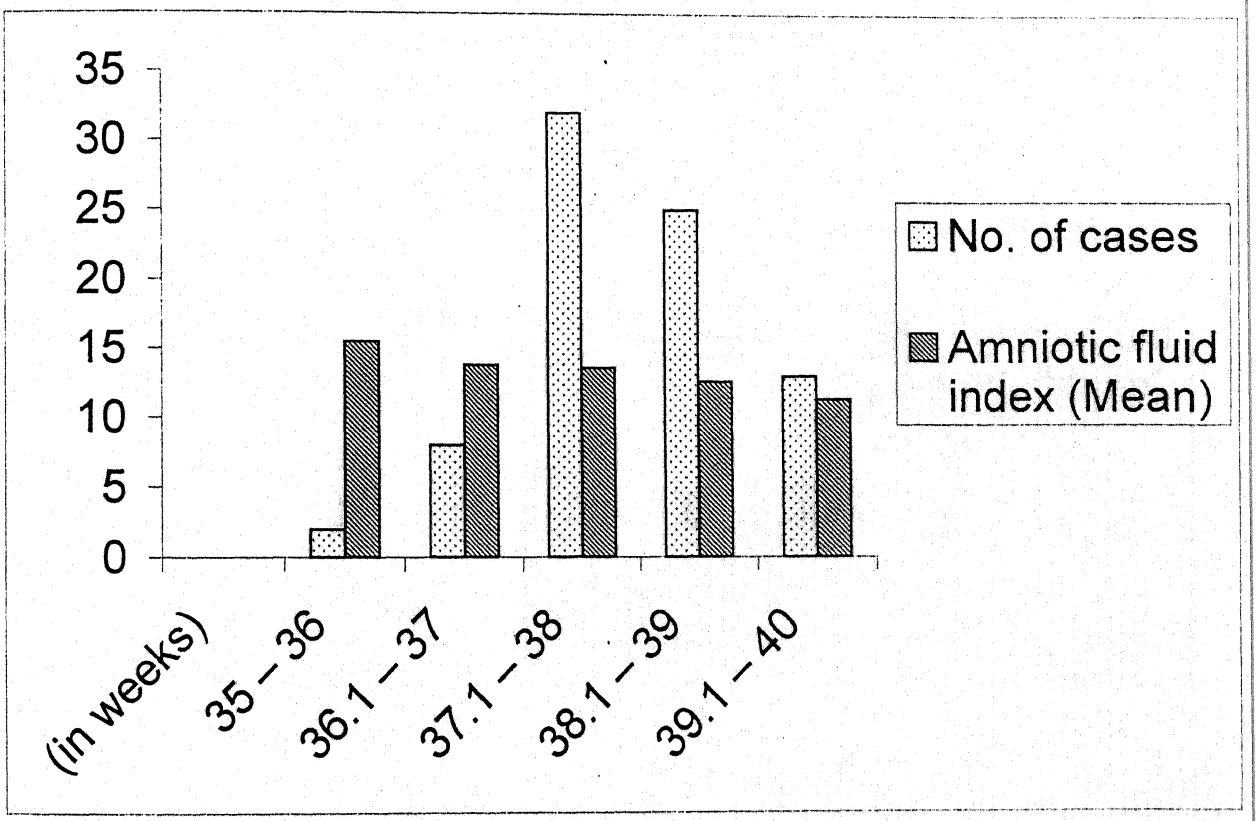
Placental grading in study group in relation to gestational age



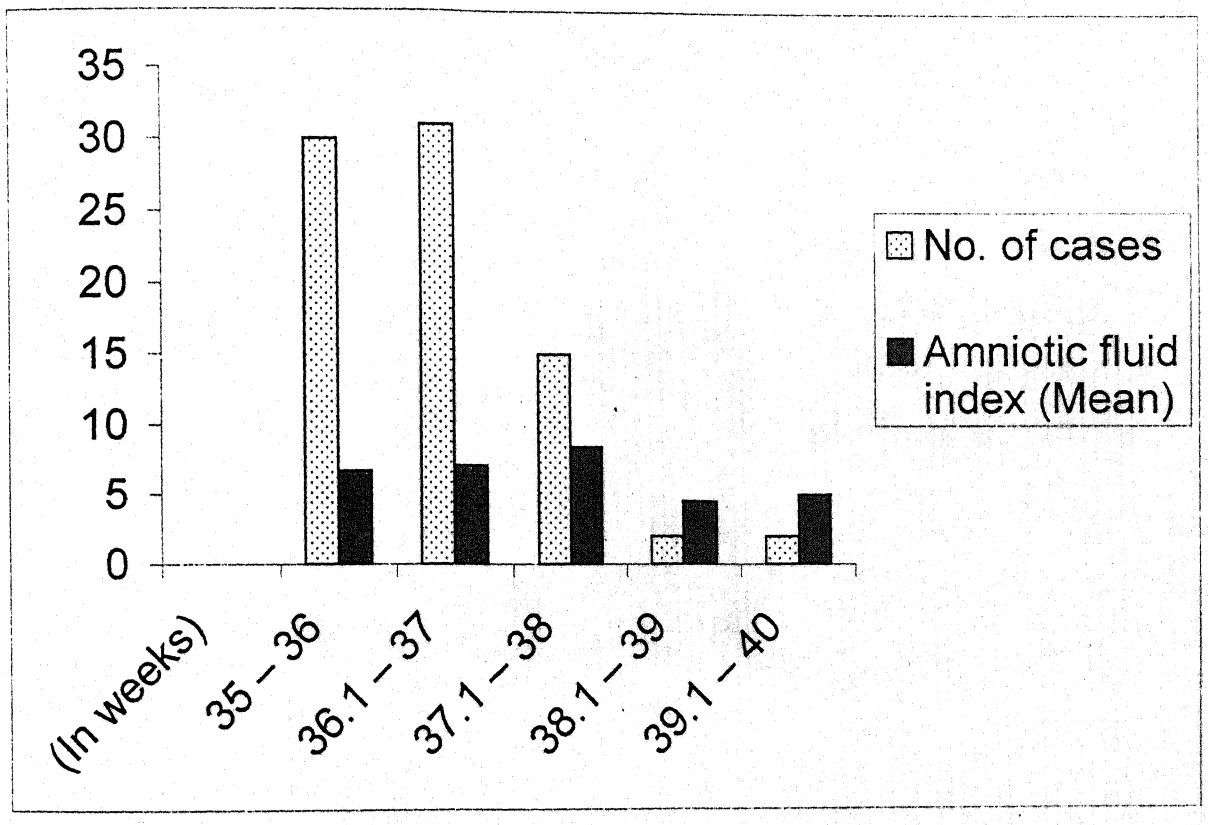
Amniotic fluid index in study and control groups



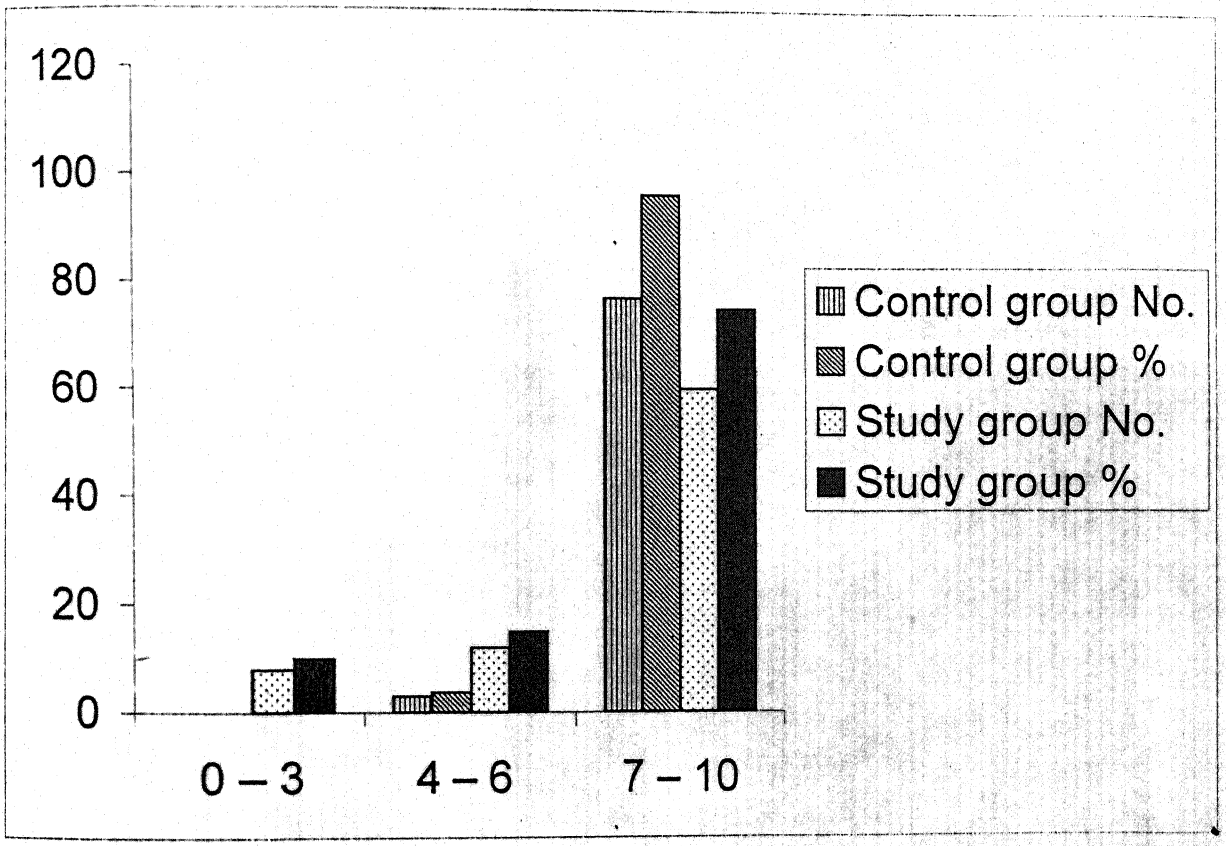
Amniotic fluid index in control group in relation to gestational age



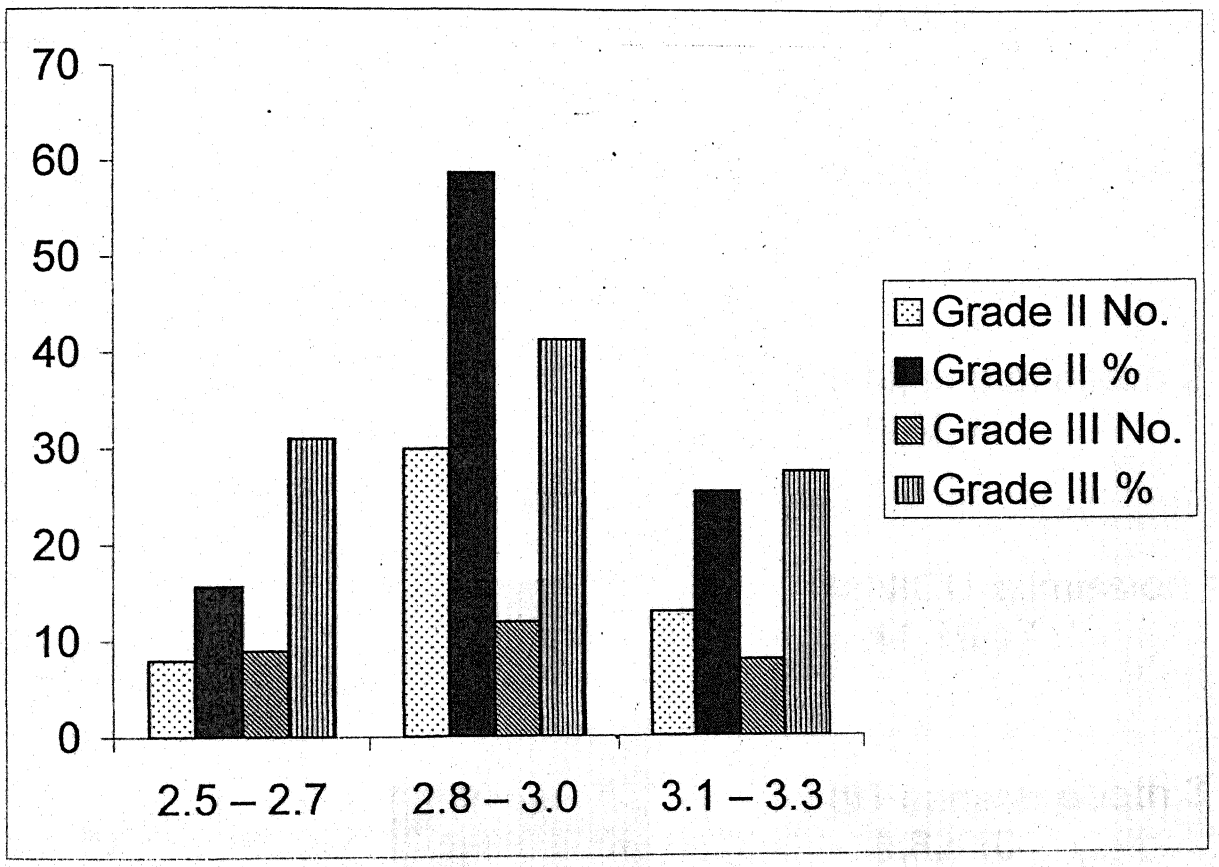
Amniotic fluid index in study group in relation to gestational age



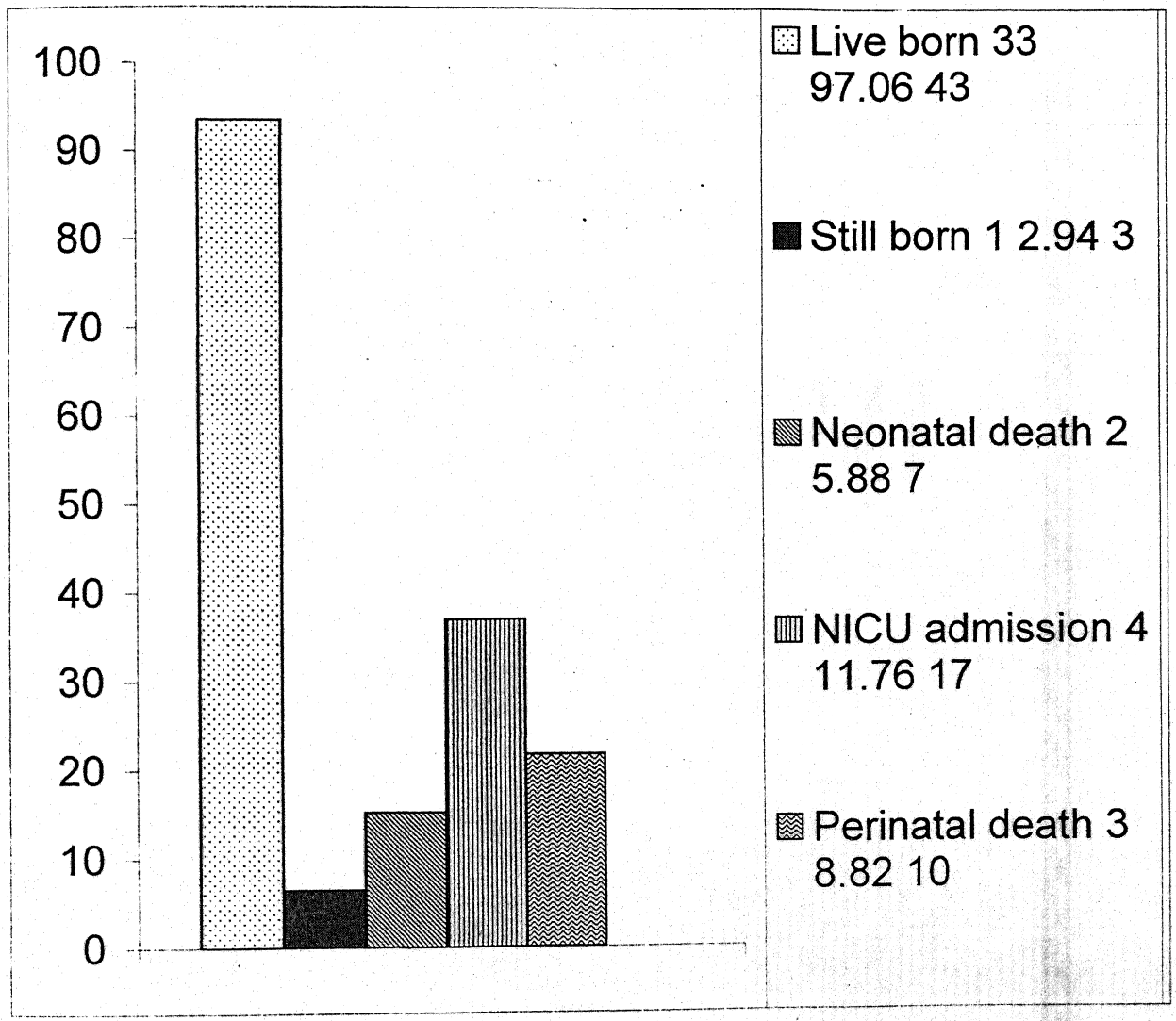
APGAR score at 1 minute



Association of placental grade and birth weight in control group



Perinatal outcome in study group in relation to placental grade



Discussion

DISCUSSION

Advances in the management of complicated antenatal situations have led to the obstetrical ethic of non-intervention and stimulated the quest for information to aid intrauterine diagnosis.

Ultrasound is becoming increasingly available in labour and delivery suites. Therefore, a simple test such as measurement of the AFI and placental grade would realistically be practical for a large number of hospitals taking very little time and providing significant amount of data.

The present study was conducted to evaluate the use of AFI and placental grading for determining the perinatal outcome in pregnancies complicated with IUGR.

The AFI was calculated by summing up the vertical dimensions of one of the largest pockets in each quadrant of the uterus. And the placental grade was determined according to the criteria of Grannum and associates with use of the most mature appearing section of placenta.

In this study 160 cases were selected. These were divided into 2 equal groups, the control and study group. The control group included the cases with 35 weeks pregnancy or more without any complications of pregnancy. The study group included patients with 35 weeks pregnancy or more and in this group in 40% of the cases antenatal complications (in 35% cases PIH was present and in 2.5% cases heart disease was present and the remaining 2.5% cases had toxoplasmosis infection) were present.

The observations were noted in terms of age, parity, period of gestation, socioeconomic status, comparison of amniotic fluid index in study and control groups and placental grading and its association with fetal outcome.

Age:

The study shows that maximum cases (73.75%) in both the groups were young between 21 – 25 years of age group i.e. 58 cases in study group and 60 cases in control group. The youngest patient was of 19 years and the oldest was of 35 years. The age was < 20 years in (13.75%) cases in study group and (11.25%) cases in control group. 4 patients (5%) in study group and 3 (3.75%) patients in control group were of 31 – 35 year age group. Both the groups were comparable regarding the age of patients, the difference was not statistically significant (P value >0.05) Table – 2.

Parity:

In the present study both primigravidae and multigravidae were included. Majority or the women in control group were primigravida (90%) and in the study group 37 (46.25%) were multigravida and 43 (53.75%) were primigravida.

Relation to rural and urban population:

More than half of the patients in both the groups i.e. 50 (62.5%) in control group and 55 (68.75%) in study group were rural dwellers. 30 cases (37.5%) in control group and 25 cases (31.25%) in study group were from urban background.

Socioeconomic Status:

The cases were allotted social classes according to modified BG Prasad classification for 1994. In the study group, majority of the patients (42; 52.5%) were in class IV and minimum number of patients (1; 1.25%) were in

class I. In control group, majority of the patients (36; 45%) were in group III, and minimum (3, 3.75%) were in class I. Class IV included (28; 35%) patients. In this study, the patients of the study group as compared to control group were from lower socioeconomic status. This shows that poor socioeconomic status is a risk factor.

A Co-relation between socio-economic status of women and the outcome of pregnancy was observed by **Gopalan** (1982) in a study of 1000 pregnancies. **Gopalan** (1982) has included social class III and IV as a high risk factor in high risk pregnancy scoring and incidence of low birth weight in the study was 22.3% and 80% perinatal deaths occurred in this group. **Jayam et al** (1984) found that a similar scoring system for screening of high risk mother was useful to lower the LBW and PNM rates.

Amount of Liquor:

Clinically in the control group 76 out of 80 cases had adequate liquor, and 4 cases had Oligohydramnios. In the study group, 67 cases out of 80 cases had Oligohydramnios and 13 cases had adequate amount of liquor. Normally amniotic fluid volume increases to about 1 litre or somewhat more by 36 weeks but decreases thereafter. Postterm there may be only a few hundred ml or even less.

Table

Typical amniotic fluid volume (from Queenan et al 1991)

Weeks (Gestation)	Foetus (gm)	Placenta (gm)	Amniotic Fluid (ml)	% Fluid
16	100	100	200	50
28	1000	200	1000	45
36	2500	400	900	24
40	3300	500	800	17

According to **Pritchard et al** (1980) Perinantal mortality and morbidity are significantly increased when oligohydramnios or polyhydramnios are associated with pregnancy.

Fetal urine contributes significantly to amniotic fluid volume from 18 weeks of gestation. Severe oligohydramnios is a common finding in bilateral renal agenesis. With diminished placental function and reduced renal perfusion the amniotic fluid volume decreases. Perinatal outcome is poor when the amniotic fluid volume is reduced at delivery. (**Chamberlain et al** 1984; C**rowly et al**, 1984).

Amniotic Fluid Index:

Clinical evaluation by abdominal palpation can be deceptive in cases with decreased fluid. Impression of the amniotic fluid volume on USG examination is fairly sensitive. Objective assessments of the vertical depth of the largest pocket of Amniotic fluid after excluding loops of cord or addition of the vertical pockets in 4 quadrants of uterus are used.

Phelan et al, 1987; **Jenu et al** 1990 Found that the measures of AFI correlated well with changes in amniotic fluid volume during the course of pregnancy and little inter and intra observer variation was found (**Rutherford et al** 1987; **Moore and Cayle**, 1990).

According to the **Moore** 1990 AFI is more sensitive in predicting fetal morbidity than the largest vertical pocket liquor assessment.

According to **Rutherford**, 1987 an AFI <5 is associated with poor fetal outcome.

In a study by **Phelan et al** (1987) in which 197 patients with good dates clinically under went 262 AFI assessment and from the study he concluded that from 11 – 26 weeks the AFI Rose progressively. Thereafter until term the AFI remained 16.2 ± 5.3cm and after 38 weeks the AFI appeared to gradually decline.

Amniotic Fluid Index Changes during Pregnancy (Phelan 1987).

Gestational Age (Weeks)	AFI (cm) + SD
34	15.7 + 4.8
36	15.9 + 4.6
38	16.1 + 6.1
40	15.3 + 6.3
42	14.1 + 3.5

Another study done in 1988 by **Cheng Jye Teng** and Colleagues for the measurement of AFI concluded –

The AFI Rose progressively from 13 weeks gestation until 26 weeks, from 26 – 38 weeks the AFI demonstrate little variation and the peak AFI appeared at 29 – 30 weeks. After 38 weeks the AFI declined progressively.

In our study amniotic fluid index in control and study group in relation to gestational age were as follows.

Gestational Age (in weeks)	Amniotic Fluid Index (mean)	
	Control Group No.	Study Group No.
35 - 36	15.5	6.7
36.1 – 37	13.8	7.1
37.1 – 38	13.6	8.4
38.1 – 39	12.6	4.5
39.1 – 40	11.3	5.0

Thus, the AFI in the control group in our study correlates with the finding of AFI changes during pregnancy as stated by **Phelan et al** (1987). There was a progressive fall in AFI with increasing gestational age although the values were slightly lower than in the study by **Phelan** (1987).

Our study was restricted to the study of AFI from 35 weeks gestational age onwards.

The results in the control group were as follows:

The mean AFI in 2 patients with 35 – 36 weeks gestational age = 15.5. There were 8 patients with gestational 36.1 – 37 weeks who had a mean AFI = 13.8. In the gestational age 37.1 – 38 weeks there were 32 patients who had mean AFI of 13.6.

In the gestational age 38.1 – 39 weeks, there were 25 cases who had mean AFI of 12.6. In the Gestational age 39.1 – 40 weeks, there were 13 patients who had mean AFI of 11.3.

In the study group:

There were 30 patients with 35 – 36 weeks gestation, who had mean AFI of 6.7. There were 31 patients with 36.1 – 37 weeks gestation, who had mean AFI = 7.1. There were 15 patients with 37.1 – 38 weeks gestation who had mean AFI = 8.4. There were 2 patients with 38.1 – 39 weeks gestation who had mean AFI = 4.5. There were 2 patients with 39.1 – 40 weeks gestation who had mean AFI = 5.

A study conducted **by SP Chauhan, Hendrex NW, Morrison JC, Magann EF, Devoe CD,** 1997 shows that oligohydramnios defined by an AFI <5cm are poor predictors of adverse outcome for high risk Intrapartum patients.

Phelan et al arbitarily defined an AFI <5cm as very low, 5.1 – 8 cm as low, 8.1 – 18 cm as normal and > 18 or 20cm as high.

Cheng Jye Jeng and associates opinion was that AFI <8cm and >24cm in the third trimester could be categorised as decreased and increased Amniotic fluid respectively. In our study we define AFI <5cm as very low, 5-8 as low; 8.1 – 18cm as normal.

In a study by **John Brein et al** (1993). He has concluded that oligohydramnios occur in a substantial proportion in patients hospitalised with hypertension.

In our study there were 28 patients of PIH and oligohydramnios. So our finding co-relate with the findings in the above study.

Statistical calculations were done and P valve was <0.01 signifying that there is a highly significant statistical difference in the AFI values in the control group and study group.

RELATIONSHIP OF AFI WITH FETAL COMPLICATIONS AND PERINATAL OUTCOME:

An association between oligohydramnios and IUGR has long been recognized and has been generally accepted.

In a study by **K Grubb B and H Paul** in which fetal outcome in relation to AFI was studied is as follows:

AF Index

	<2cm	2 – 4 cm	>5cm
	(N = 11)	(N = 17)	(N = 64)
Intervention for Fetal distress	7(64%)	4(24%)	13(20%)
Caesarean Section for Fetal Distress	6(55%0	3(18%)	9(14%)
Meconium Passage	7(64%)	6(35%)	13(20%)

They concluded that intervention for fetal distress was required in 27% of patients. The rate of intervention was significantly higher in women with AFI <2cm than those with values between 2–4.9cm or those with Index of 5cm or >.

The fetus passed Meconium before delivery in 29% of cases. The incidence of meconium passage was significantly higher in patients with low AFI.

In our study of 80 patients in the study group 67 (83.75%) patients had reduced AFI.

Meconium staining of liquor was seen in 18 patients out of 67 patients with reduces AFI i.e. 26.87% patients.

However, in the control group meconium staining of liquor was seen in one patient out of 4 patients with reduced AFI.

These findings shows that the incidence of meconium passage is higher in cases with altered AFI.

A study was carried out by **SP Chauhan, M. Sanderson, NW Hendrix, EF Magnann, CD Devoe** (1999). To find the risk of Caesarean delivery for fetal distress, 5 minutes apgar score <7 and umbilical artery pH <7:00 in patients with antepartum or Intrapartum AFI < 5.0.

The conclusion of the study was that an antepartum or intrapartum AFI ≤5.0cm is associated with a significantly increased risk of caesarean delivery for fetal distress and a low apgar score at 5 minute.

In our study in the study group out of 80 patients, 20 patients had an AFI <5cm. Out of these 20 patients, 12 patients had undergone a caesarean section i.e. 60%.

The rate of operative intervention was much higher in the study group and 30 patients i.e. 37.5% had to undergo lower segment caesarean section as compared to 5% patients in control group.

In the control group, there were three patients with Apgar score <7/10 at 1 minute. In the study group, there were 20 patients in the apgar score <7/10 at 1 minute, and 10 patients had an apgar score <7/10 at 5 minutes.

According to a study carried out by **EH Banks** and **DA Miller,** 1999, there was a 2 fold increase in the incidence of adverse perinatal outcome

among the women with borderline AFI in comparison with control subjects with normal AFI. There was a 4 fold increase in the incidence of fetal growth restriction among women with borderline AFI.

In our study statistical calculations were done and it was found that there is a significant difference in the apgar score at 1 minute in the control group and study group. i.e. $P<0.05$.

In the present study:

The cases were followed and there was no perinatal mortality in the control group. In the study group, there was no perinatal mortality in patients with normal AFI.

There were 7 perinatal deaths out of 67 cases with reduced AFI. In these 7 cases, PIH was associated with IUGR, thus signifying that reduced AFI is an indicator of Intrauterine Fetal stress and reduced placental perfusion.

Placental Grading:

In our study of 80 patients in the study group 34 patients (42.5%) had grade II placental changes and 46 patients (57.5%) patients had grade III placental changes. Whereas in control group, 51 (63.75%) patients had grade II placental changes and 29 patients (36.25%) had grade III placenta changes. Incidence of grade III placenta was high in the study group as compared to control group.

Relationship Of Placental Grade With Perinatal Outcome:

In our study, in the study group, the fetal distress was present in 22 patients (64.71%) in grade II and 31 patients (67.39%) in grade III placenta. Whereas, in control group fetal distress was present in 3 (5.88%) patients in

grade II placenta, and in 2 patients in (6.9%) in grade III placenta. This shows that incidence of fetal distress was more in study group than in control group. However, the incidence of fetal distress was similar in grade II and grade III placentae in both the groups.

In our study, in the study group, the mean birth weight in patients with grade II placental changes was 2.056 ± 0.47kg and the mean birth weight in patients with grade III placental changes was 1.878 ± 0.22 kg which was significantly lower as compared to grade II placenta ($P < 0.05$).

In control group, the mean birth weight in patients with grade II placental changes was 2.929 ± 0.2 kg and with grade III placental changes the mean birth weight was 2.889 ± 0.024 kg, which was slightly less than grade II. This difference was not statistically significant ($P > 0.05$).

In the study group, 76 babies (95%) were live born, 4 (5%) babies were still born. 9 babies (11.25%) died in the neonatal period. There was no such adverse perinatal complications found in control group. 21 babies out of 80 in study group were admitted in NICU where as in control group only 2 babies were admitted.

In the study group, in grade II placenta, 33 babies (97.06%) were live born, and 1 baby (2.94%) was still born. 3 babies (8.82%) died in the neonatal period, 4 babies (11.7%) were admitted to NICU.

In grade III placenta, 43 babies (93.48%) were live born and 3 babies (6.52%) were stillborn. 7 babies died (15.2%) in the neonatal period and 17 babies (36.96%) were admitted in NICU.

In the present study:

The grade III placenta in study group was associated with lower birth weight, increased incidence of admission to neonatal intensive care unit and increased perinatal mortality.

In the control group, placental grading had no correlation with perinatal outcome. The growth retarded fetuses had higher incidence of grade III placenta which is associated with poor perinatal outcome.

The relationship of placental grade with birth weight and perinatal outcome in pregnancies complicated with IUGR as found by different authors in different studies is:-

1. **Ruth A. Petrucha and Lawrence D. Platt (1982):** In a study on 964 patients to find the relationship of placental grade to gestational age found that in each of four cases where a grade III placenta occurred before 35 weeks, intrauterine growth retardation was found in the fetus.

2. **Robert M. Patterson, Robert H. Hayashi, and Dora Cavazos (1983):** Carried out a study on 398 patients, to find out the relationship of early placental maturation and perinatal outcome. The total population of 398 patients contained 21 infants defined as SGA (5.3%). They found that in the grade II population, the incidences of SGA were 9.5% and 4.0% in the early placental maturation and control patients, respectively. In the grade III population, SGA occurred in 16.7% of the patients with early placental maturation and in 4.1% of the control patients, a significantly increased incidence ($P<0.01$). The sensitivity of early placental maturation as a marker for SGA infants was 46% with a predictive values of 16.7%.

3. **George M. Kazzi; Thomas L. Gross; Robert J Sokol, Nadya J. Kazz, (1983):** Carried out a study on 224 patients who were examined sonographically with in 7 days of delivery. Of these, 109 pregnancies resulted in birth of small infants who weighed ≤ 2700gm. Of the 109 patients, 44 had grade III placentas and 65 had non grade III (0, I, II) placentas. They found that the mean birth weight percentile was significantly less in the grade III placenta group than in the non grade III group. ($P < 0.0001$).

4. **Grannum and Hobbins (1979):** At term, in normal patients, they found 40% grade I placenta, about 40% grade II placenta and 15 – 20% grade III placenta. IUGR was seen with grade I placenta in 4% of cases, with grade II placenta in 19.7% and with grade III placenta in 26.3% of the cases.

5. **Fisher et al (1976)** and **Petrucha et al** (1982) showed that premature senescence of placenta was associated with IUGR.

6. **Quinlan** et al (1982) and **Tabsch** (1983) reported that preterm appearance of grade III placenta was associated with adverse perinatal outcome.

7. **Kamla Ganesh, Uma Dhawan and N.C. Gupta (1989):** Carried out a study on 140 patients, with study group having 100 primigravidae with PIH and 40 control with normotensive pregnancy. They were studied for placental grading by ultrasound and the findings were correlated with foetal outcome. They found that IUGR babies resulted in 7.1% of the cases of PIH with grade I and 23.2% with grade II and 31.3% with grade III placenta. Foetal distress was significantly more with grade III placenta ($P< 0.01$). The incidence of perinatal deaths in patients with grade II and III placenta was 3.6% and 12.5% respectively.

8. **Veena Agrawal, Sapna Jain (2000):** Found that in hypertensive and IUGR cases placental maturity was accelerated being higher than in normal pregnancy of similar gestational age. They observed that if placenta maturates early i.e grade III placenta before 37 weeks, fetal complication occurs in the form of IUGR, fetal distress and neonatal death.

9. **Quinlan** and Associates, (1982): Described 41 patients who had grade III placentas prior to term. They found a significant incidence of maternal hypertension and fetal growth retardation and suggested that early placental maturation might predict complications in late pregnancy.

10. **Sanjay Kumari, Harjeet Sawhney, Kala Vasishta, Anil Narang** (2001): Carried out a study on 100 pregnant patients with a study group comprising of 50 pregnant patients with IUGR and a control group of 50 patients with fetal growth appropriate for gestational age. They found that the incidence of grade III placenta was significantly high in the study group as compared to control group ($P < 0.05$). Placental grading had no correlation with incidence of fetal distress and meconium stained liquor in both the groups. Birth weight was significantly lower in grade III placenta (1482.3 ± 320.5g). as compared to grade II placenta (1766 ± 484.7gm) in growth retarded fetus.

Summary & Conclusion

SUMMARY AND CONCLUSIONS

The present study was carried out in the department of obstetrics and gynaecology – Maharani Laxmi Bai Medical College, Jhansi.

The aim of study was to study the effect of placental grading and amniotic fluid index in IUGR and to correlate these with fetal outcome.

A total of 160 cases were taken, 80 in the control and 80 in the study group.

Control Group:

80 antenatal patients with 35 weeks of pregnancy or more without any complications of pregnancy and with fetal growth appropriate for gestational age were included.

Study Group:

80 antenatal patients with 35 weeks of pregnancy or more with one or more antenatal complications and with fetal growth small for gestational age were included.

On the basis of our study the following conclusions were made:

1. The age distribution of patients varied from 18 – 35 years. Maximum number of cases were young and belonged to 21 – 25 years of age group (73.75%). The mean age in the control and study groups were 23.48 year and 23.46 year respectively.

2. There were 90% primigravida in control group and 43% primigravida in the study group.

3. Majority of the women in study group had weight less than 56kg and in control group majority had weight >55kg. The mean maternal weight in the control and study groups were 55.60kg and 54.88kg respectively.

4. More than half of the patients in both the groups were rural dwellers. In study group 55 (68.75%) patients were from rural background and 25 (31.25%) patients were from Urban background. In the control group, 50 (62.5%) patients were from rural background and 30 (37.5%) patients were from Urban background.

5. More than half of cases were either illiterate or had only primary education, i.e. 62.5% in study group and 55% in control group. Rest were educated upto high school, intermediate or were graduate.

6. In the study group, majority of the patients (42; 52.5%) were in social class IV and minimum number of patients were in class I. In control group majority of the patients (36; 45%) were in social class III and minimum were in class I.

7. In the study group 32 (40%) women had antenatal complication, in the present pregnancy (28 patients had PIH; 2 patient had heart disease; and 2 patient had Toxoplasmosis). While in control group, all pregnancies were uncomplicated.

8. In the study group 34 (42.5%) patients had grade II placental changes and 46 (57.5%) patients had grade III placental changes. Where as in

control group 51 (63.75%) patients had grade II placental changes and 29 (36.25%) patients had grade III placental changes. Incidence of grade III placenta was high in the study group as compared to control group.

9. In control group, the mean gestational age in grade II placenta was 38.03 ± 0.3 weeks and in grade III placenta was 38.07 ± 0.83 weeks.

In study group, the mean gestational age in grade II placenta was 36.50 ± 1.05 weeks and in grade III placenta was 36.44 ± 1.09.

10. AFI was calculated in all the patients of both the groups. In control group, most of the patients i.e. 76 (95%) patients had AFI ranging from 8.1 – 17cm, and 4 patients (5%) had AFI ≤ 8cm.

In the study group, 20 (25%) patient had AFI ≤ 5 cm. 47 (58.7%) patients had AFI ranging from 5.1 - 8cm, and 14 patients had AFI panging from 8.1 – 14cm.

Statistical calculations were done and P value was < 0.01 signifying that there is a highly significant statistical difference in the AFI values in the control and study group.

11. Mean AFI in relation to the gestational age in the control group was found to be 15.5cm in cases with gestational age 35 – 36 weeks, 13.8 cm in cases with gestational age 36.1 – 37 weeks, 13.6 cm in cases with gestational age 37.1 – 38 weeks, 12.6 cm in cases with gestational age 38.1 – 39 weeks, 11.3 cm in cases with gestational age 39.1 – 40 weeks.

The AFI was found to gradually decrease with increasing gestational age.

12. In study group mean AFI in patients with gestational age was: There were 30 patients with 35 – 36 weeks gestation who had mean AFI of 6.7cm. The mean AFI was 7.1cm in cases with gestational age 36.1 – 37 weeks, 8.4cm in cases with gestational age 37.1 – 38 weeks, 4.5cm in cases with gestational age 38.1 – 39 weeks, 5 cm in cases with gestational age 39.1 – 40 weeks.

13. In control group, fetal distress was present in 3 (5.88%) patients in grade II placenta, and in 2 patients (6.9%) in grade III placenta. The incidence of fetal distress in labour was similar in grade II and grade III placenta.

14. In study group, fetal distress was present in 22 patients (64.71%) in grade II placenta and 31 patients (67.39%) in grade III placenta. The incidence of fetal distress was similar in grade II and grade III placenta. The incidence of fetal distress was more in study group than in control group.

15. 95% patients in the control group had a normal vaginal delivery and caesarean section was done in 5% or patients. In study group, normal vaginal delivery occurred in 62.5% of patients and caesarean section was done in 37.5% patients.

16. The apgar score at 1 minute in the control group was between 7 – 10 in 96.25% of cases. It was between 4 – 6 in 3.75% of cases. In the study

group, the apgar score was between 0 – 3 in 10% of cases, between 4 – 6 in 15% of cases and between 7 – 10 in 75% of cases.

17. None of the babies in control group had an apgar score of < 7 at 5 minute. In the study group 12.50% babies had apgar score of < 7 at 5 minute. The apgar score was between 7 – 10 in 87.5% of babies at 5 minutes in study group.

18. In the study group, 95% babies were live born and 5% of babies were still born. 11.25% babies died in the neonatal period. There was no perinatal deaths in the control group.

19. In the study group, there was no perinatal mortality in patients with normal AFI. There were 7 perinatal deaths out of 67 cases with reduced AFI. In these 7 cases, PIH was associated with IUGR, thus showing that reduced AFI is an indicator of intrauterine fetal stress and reduced placental perfusion.

20. In study group, in grade II placenta, 97.06% of babies were live born and 2.94% was still born. 8.82% of babies died in the neonatal period, and 4 babies were admitted to NICU.

In grade III placenta, in study group, 93.48% babies were live born and 6.52% babies were stillborn. 15.22% babies died in the neonatal period and 36.96% babies were admitted in NICU.

CONCLUSION

1. In the present study, the incidence of grade III placentae was higher in growth retarded fetuses compared to the group where fetuses were appropriate for gestational age. The grade III placenta had no correlation with incidence of meconium staining of liquor and fetal distress in labour. However, grade III placenta was associated with lower birth weight, increased incidence of admission in neonatal intensive care unit and increased perinatal mortality in the growth retarded fetuses. In conclusion growth retarded fetuses had higher incidence of grade III placenta which is associated with poor perinatal outcome.

2. Our study confirmed the usefulness of amniotic fluid index measurement as a tool for evaluation of foetal well being. It is a simple and useful procedure and can be used as a screening method in antenatal period in high risk pregnancies as it is a reasonable means of assessing the fetal condition throughout pregnancy. Ultrasonographic estimation of amniotic fluid volume by AFI is a highly sensitive method and has a significant role in predicting fetal complications, as reduced amount of liquor reflects decreased fetal contribution to the amniotic pool as a result of chronic intrauterine stress and is thus associated with poor fetal outcome. Oligohydramnios and decreased volumes are highly accurate in predicting fetal distress in labour and low apgar scores in the newborn babies.

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